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# The Manitoba Medical Review

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## Symposium on Trauma

### Automobile Trauma on Manitoba Highways

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Part 1

Trauma is no respecter of time, place or person, and there is little doubt that the home and its immediate environment is, and probably will always be, the prime producer. Industry, highway traffic, and, increasingly of late, water accidents are in that order, the next important sources.

The resultant severity of the trauma is usually, however, in an inverse ratio. Water accidents are largely fatal. Highway traffic produces many fatal and non-fatal serious injuries. Industry and the home produce less severe trauma and a lower ratio of deaths.

Statistics for highway traffic accidents and fatalities show the following appalling totals for the whole of Canada:

| | 1958 | 1959 |
|---|---|---|
| Accidents | 227,451 | 241,685 |
| Killed | 3,118 | 3,213 |
| Injured | 80,061 | 84,374 |

The Province of Manitoba produced the following figures for the first ten months of 1959 and 1960.

| | 1959 | 1960 |
|---|---|---|
| Accidents | 10,237 | 10,006 |
| Killed | 127 | 99 |
| Injured | 3,954 | 3,832 |

The slight decrease in 1960 figures is further enhanced by the knowledge that there was an increase of 7% in the number of motor vehicles registered. Nevertheless, this presents a formidable crop of which we cannot be proud.

A glimpse of other countries shows the problem to be world wide and of increasing social importance. In 1959, Great Britain had 6,500 highway traffic deaths, West Europe 37,000, U.S.A. 37,000, and Victoria, Australia, reported the worst mortality rate in the world for that year. The overall proportion of dead to injured is approximately 1:100.

The handling of highway traffic accidents poses many interesting problems in Manitoba, which of course, are modified to some extent by local conditions.

In Urban areas, the immediate situation is usually dealt with by ambulance crews and "First Aiders." Not so in the rural areas, where the higher speed of vehicles, resulting in more serious injuries, requires a medical man on the scene. Indeed it is getting so that no one will move injury victims until they have been medically examined. Most of us will agree that the closer we work to the scene of trauma, the more lives we will save.

The alarm of an accident is usually received by the police first, and while they dispatch their nearest patrolling car by short wave radio, a medical man and ambulance are summoned. The police have traffic controlled or deviated by the time rescue units are present. This organized team work seems to work out very well, and, with everyone knowing his particular job, little time is lost in evacuating the victims. The much discussed use of police station wagons for this purpose has no place in this scheme. The axioms of thoroughness, common sense, good judgment and decisive action then come into play, and the medical man is looked upon as a leader, and his professional judgment is rarely questioned.

Some preparation for these events is necessary. Tracheostomy sets, splints, drugs, dextran or plasma, blankets, good light (many of these accidents occur after dark), baggage tags and oxygen tanks are among the many items carried. Adequate personal clothing, especially rubber boots and a camera should be among the high priority items.

At the scene of a hypothetical accident, a quick general survey is made of the locale with particular reference to the number injured. Many times a victim may be several hundred yards away from the main wreckage. Some do this by jumping from the moving vehicle which is going out of control, and some wander away in a stupor and collapse.

Righting an overturned vehicle with people trapped in it can be a problem, but five people with a will and in unison can right an upset vehicle. Releasing wedged victims, especially the driver, calls for a good deal of ingenuity. An ordinary bumper jack, strategically propped against a door post, can move the whole front of the car sufficiently to extricate a victim from behind the steering wheel. An axe can be used to break seat frames or cut door posts. At times an acetylene cutting torch is required.

Striking and knocking down power line poles is fairly common now-a-days, and as much of the power is transmitted in rural areas at 550 volts without benefit of insulation, it is a hazard to victim and rescuer alike. Thanks to short wave radio it is a simple matter to get the power shut off, but all too often it is too late for some "Would-be Helper."

A rapid superficial examination of all the victims, usually with clothes on, is made, and a decision reached as to who requires immediate further care. This triage or sorting is the key to the successful

medical management of a disaster or accident, no matter what its magnitude. First things come first. Establishing an adequate airway takes precedence and one should not hesitate to do an immediate tracheostomy, no matter where you are. It should be done when it is first suspected one may be necessary, rather than wait until it is absolutely essential. The newer types of apparatus available make this a relatively simple procedure, with little danger of subsequent laryngeal stenosis. A No. 13 gauge needle may suffice, especially in children. An ordinary safety pin through the tongue, and held or attached to clothing is often sufficient for transport.

Gross hemorrhage next commands attention and it is surprising how many are controlled by simple pressure dressings. Tourniquets should only be applied if a strict time limit is adhered to. A maximum of thirty minutes is safe, and if it appears unlikely you can attend to it then, more time must be spent to clamp and tie vessels on the spot. Perhaps because of this low maximum time limit "Tourniquet Shock," described by Royster of Philadelphia, will rarely be seen, in spite of multiple soft tissue injuries.

Shock with all its lethal factors, next commands attention and of prime importance is adequate external heat production. There is room for some inventive genius to produce an electric or chemically heated blanket for use at accident scenes. Even in warm weather the patient lying exposed and in shock is losing vitality rapidly. In zero or below weather many lives are lost which warmth in adequate amounts would save. Wound shock is essentially hypovolemic, and loss of blood volume is in most cases due to hemorrhage. The immediate ideal primary treatment of shock due to injury is treatment of the wound itself and replacement of blood. Before these ideals are accomplished ordinary measures go far in preserving hemostasis and allowing the reflex physiological mechanisms time to act, by means of compensatory fluid shifts. These factors can be temporarily bolstered by artificial volume expanders or plasma. Dextran at the roadside can be a real life saver. It does not freeze until zero is approached, but one might well question the value of pouring a chill liquid into a shocked victim without adequate external heat.

Fractures present the next problem. With careful handling many of them can be transported without splinting, although if the limb is grossly shattered, time must be taken to establish temporary splinting. Flail chests and sucking wounds can be temporarily helped by mild pressure dressings or by safety pins or towel clips attached by rubber bands to the roof or wall of a vehicle.

The use of narcotics is a controversial point. Intravenous morphine is certainly the speediest relief to suffering, but should never be given in cases of head injury or indeterminate visceral injuries. Codeine by hypo may be resorted to for severe pain in such cases. An ordinary tie-on baggage label offers a convenient record of what and when it was given. On this may also be noted the most expedient disposition of the patient on reaching the hospital. Lacking a suitable label, the information may be written on the forehead.

Transporting the victim seldom offers a problem except in the case of neck or back injuries. The so-called "coma" or lateral position is probably safer whenever there is any doubt. Traction on the neck can be maintained during transport. Primary damage may be compounded by bad emergency treatment.

The ambulance having been sped on its way, a quick radio call alerts the hospital as to what will likely be needed in the way of services. Before leaving the scene of an accident, it is invaluable to have a word with other uninjured or mobile occupants of the vehicle. Frequently they can tell of chronic sicknesses, or current medications, of the other victims. This applies particularly to cardiacs, diabetics, victims of renal disease, and epileptics. Tuberculosis is still with us and must be constantly watched for. The freer use of steroids has recently added another problem to the list.

Admission to hospital can be a hazard to the critically injured and a few notes on the previously mentioned humble baggage tag minimizes the mislaying of people in a labyrinth of corridors and odd corners where they may be pushed pending attention to something more urgent.

Victims should be admitted directly to emergency service bed, X-ray department, or emergency operating room. Wherever they are, their clothing is cut or removed, and a thorough examination and evaluation of injuries made to expedite definitive treatment.

Airway, shock and hemorrhage must always take precedence as at the actual scene. We are being forced to accept artificial volume expanders due to lack of readily available blood, and it must be admitted (grudgingly perhaps) the results are fairly satisfactory. Blood studies from the laboratory seem of little value in early trauma, and one is forced to rely on clinical judgment. Early establishment of vital signs and records is essential, and once again the baggage tag plays a role.

Emergency X-ray work in the X-ray unit should be limited to the so-called "Vital X-rays," that is, chest, skull and abdomen. Few X-ray units are equipped to care for concomitant emergencies. Portable units are frequently satisfactory for extremities, or they may be delayed until emergency pressures are over, as can considerations such as antibiotics, vaccines, catheterization, fluid balance and nourishment.

As a medico, it is frequently necessary to return to the scene of an accident, after the victims have been cared for. There, measurements and photographs are being carefully taken and tabulated.

Witnesses are interviewed and all preliminary work completed, which a year or two later, will be dissected in litigation proceedings. Shortly thereafter tow trucks appear and in a few minutes the scene is back to normal and largely forgotten.

Observing this routine time after time soon generates the usual "Why." What mechanisms and forces are at work to produce similar types of injury, and what can be done to stop or minimize this wholesale dissolution and damage to human beings on the highway?

It represents our most pressing problem in terms of the loss of useful and productive years of life. Recognition of the automobile as being as great an environmental hazard as bacteria is long overdue. By our inaction we show surprising apathy and lethargy in setting an example of effective leadership in preventative medicine. This problem requires the same strategic approach as in eradicating any epidemic.

The keynote, as in every other field, lies in education, and the strict enforcement of acceptable rules and laws. The police investigate from the viewpoint of law breaking. The insurance investigates to determine negligence. Investigation of the human element, predominantly responsible for 95% of the carnage, must fall on the shoulders of the medical profession. A vast amount of knowledge in this field has been accumulated, but is not effectively applied.

The problem resolves itself into one of the following categories:

1) Driver hazard
2) Speed of vehicles
3) Highway conditions
4) Equipment.

1) "A man drives as he lives," characteristics including egocentricity, aggressiveness, anti-social trends and social irresponsibility, lead to the development of the "accident prone" driver. Private car drivers under 30 show the highest accident rate, irrespective of experience, while the occasional inexperienced driver doubles the road hazard. Table 1 shows how consistent this age factor has been, over the past three years. It further shows each of us what our accident potential is. Table 2 shows license suspensions for all driving infractions (including accidents), and it is of interest to note the 16-19 year old group shows a better performance than the 20 to 34 year old group. This may well indicate the early dividends on better driver training programmes.

**TABLE 1**
**PROVINCE WIDE STATISTICS**
**A Breakdown of Drivers Involved in Accidents by Age Group in 1959**
**and**
**A Breakdown of All Licensed Drivers by Age Group in 1959 Followed by the Involvement Rate Per 100 Drivers**

| Age of Driver | No. of Drivers Involved In Each Age Group | % of Drivers Involved In Each Age Group | Total No. Drivers 1955 | 1956 | 1957 | 1958 | 1959 | % of Total Licensed Drivers In Each Age Group 1959 | 1955 | 1956 | 1957 | 1958 | 1959 | 1959 over 1955 Percent Change |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 16-19 | 2,387 | 10.4% | 21,924 | 21,406 | 22,007 | 22,850 | 24,662 | 7.1% | (4) 7.4 | (6) 8.2 | (8) 9.2 | (7) 8.8 | (7) 9.7 | +31.1% |
| 20-24 | 4,118 | 17.9% | 38,714 | 38,714 | 35,993 | 41,377 | 42,652 | 12.4% | (8) 9.8 | (7) 10.4 | (7) 8.5 | (8) 9.2 | (7) 9.7 | —1.0% |
| 25-34 | 6,497 | 28.2% | 77,739 | 76,116 | 87,644 | 81,495 | 88,746 | 25.8% | (7) 8.8 | (6) 8.2 | (6) 6.2 | (6) 7.0 | (6) 7.3 | —17.0% |
| 35-44 | 4,671 | 20.3% | 62,295 | 67,873 | 75,602 | 74,065 | 79,217 | 23.0% | (5) 7.8 | (5) 6.4 | (5) 4.9 | (5) 5.5 | (5) 5.9 | —24.4% |
| 45-54 | 3,195 | 13.9% | 37,019 | 46,326 | 51,378 | 51,962 | 57,418 | 16.7% | (6) 7.9 | (4) 5.6 | (4) 4.6 | (4) 5.1 | (4) 5.6 | —29.1% |
| 55-64 | 1,479 | 6.4% | 21,921 | 25,698 | 29,099 | 29,609 | 32,014 | 9.3% | (3) 6.8 | (2) 5.1 | (3) 4.1 | (3) 4.4 | (3) 4.6 | —32.4% |
| 65-74 | 577 | 2.5% | 9,189 | 10,399 | 14,436 | 14,431 | 15,734 | 4.6% | (2) 4.3 | (3) 5.3 | (2) 3.4 | (2) 3.5 | (2) 3.7 | —1.4% |
| 75 and over | 91 | .4% | 2,145 | 2,103 | 3,198 | 3,171 | 3,884 | 1.1% | (1) 3.8 | (1) 2.7 | (1) 2.2 | (1) 2.9 | (1) 2.3 | —39.5% |
| Sub-Totals | 23,015 | 100.0% | 270,946 | 285,914 | 326,218 | 316,418 | 344,327 | 100.0% | 8.2 | 7.2 | 5.8 | 5.8 | 6.7 | —18.3% |
| Not Stated | 320 | | 904 | 1,122 | 1,477 | 1,305 | 960 | | | | | | | |
| GRAND TOTALS | 23,335 | | 271,850 | 287,036 | 326,218 | 317,723 | 345,287 | | | | | | | |

**TABLE 2**
**ANALYSIS OF DRIVERS SUSPENDED BY AGE GROUPS, 1957, 1958, 1959**

| Age of Driver | No. of Drivers Suspended 1957 | 1958 | 1959 | Percent of Drivers Suspended in Each Age Group 1957 | 1958 | 1959 | No. of Drivers in Each Age Group 1957 | 1958 | 1959 | Percent of Drivers Each Age Group 1957 | 1958 | 1959 | No. of Drivers Suspended per 1,000 1957 | 1958 | 1959 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 16-19 | 570 | 592 | 707 | 5.7 | 5.7 | 5.4 | 22,007 | 22,850 | 24,662 | 6.7 | 7.2 | 7.1 | 25.9 | 25.9 | 28.6 |
| 20-24 | 1,880 | 1,945 | 2,490 | 18.8 | 18.9 | 18.9 | 41,377 | 38,787 | 42,652 | 12.7 | 12.2 | 12.4 | 45.4 | 50.1 | 58.4 |
| 25-34 | 3,100 | 3,178 | 4,091 | 31.0 | 30.8 | 31.1 | 87,644 | 81,495 | 88,746 | 26.9 | 25.7 | 25.7 | 35.4 | 38.9 | 46.1 |
| 35-44 | 1,880 | 2,046 | 2,585 | 18.8 | 19.8 | 19.7 | 75,602 | 74,065 | 79,217 | 23.2 | 23.3 | 22.9 | 24.8 | 27.6 | 32.6 |
| 45-54 | 1,140 | 1,169 | 1,456 | 11.4 | 11.4 | 11.1 | 51,378 | 51,962 | 57,418 | 15.7 | 16.4 | 16.6 | 22.2 | 22.4 | 25.4 |
| 55-64 | 600 | 592 | 771 | 6.0 | 5.7 | 5.9 | 29,099 | 29,609 | 32,014 | 8.9 | 9.3 | 9.3 | 20.6 | 19.9 | 24.1 |
| 65-74 | 390 | 377 | 469 | 3.8 | 3.6 | 3.6 | 14,436 | 14,431 | 15,734 | 4.4 | 4.5 | 4.6 | 26.3 | 26.1 | 29.8 |
| 75 and up | 150 | 202 | 303 | 1.6 | 1.9 | 2.3 | 3,198 | 3,171 | 3,884 | 1.0 | 1.0 | 1.1 | 5.00 | 63.7 | 78.1 |
| Not Stated | 183 | 231 | 264 | 2.9 | 2.2 | 2.0 | 1,477 | 1,305 | 960 | 0.5 | 0.4 | .3 | 30.3 | 32.5 | 38.0 |
| Total | 9,893 | 10,332 | 13,136 | | | | 326,218 | 317,693 | 345,287 | | | | | | |

| | Total Licensed Drivers 1958 | 1959 | Percentage of Drivers by Sex 1958 | 1959 | Percentage of Drivers Suspended by Sex 1958 | 1959 |
|---|---|---|---|---|---|---|
| Male | 239,358 | 258,015 | 75.3 | 74.7 | 90.3 | 89.1 |
| Female | 76,153 | 87,272 | 24.0 | 25.3 | 7.1 | 10.9 |
| Not Stated | 2,182 | 0 | 0.7 | 0% | 3.6 | 0% |
| | 317,693 | 345,287 | | | | |

Anticipation of accident situations and their careful avoidance are more important driver qualities than fast reaction times. Fatigue plays an all important role in both of course, and inattention and drowsiness are cardinal symptoms. Other stresses, such as temperatures below 50 degrees, or above 87 degrees, if sustained, impair efficiency.

The use of drugs such as antihistamines, hypnotics, sedatives and ganglionic blocking agents are all contributing factors to lower driving standards, inducing lack of concentration and drowsiness. The amphetamines temporarily increase alertness and efficiency, but may also induce irritability and finally a compensatory depression.

Axiomatic! "A man drives his best when he has had no alcoholic drinks." (Neurosurgeon G. F. Rowbotham).

Sufficient experimental and statistical data have been documented to show that small amounts of alcohol have a measurable detrimental effect on driving skill. Impairment of driving ability parallels the blood and urine alcohol levels. Compulsory blood tests are a legislative rather than a medical problem, but should be instituted when we stop to digest the fact that alcohol is a factor in 80% of our Provincial fatal accidents. Impairment begins with as low a level as 20-30 mg. per 100 ml. blood. This, of course, is subclinical and not a significant cause of accidents. At and above 100 mg. per 100 ml. blood, it may be of importance as a factor in driving. Conditions for impaired driving, in other countries, show wide variations.

| | | |
|---|---|---|
| Norway | 80 mg. | per 100 ml. blood |
| Sweden | 100 mg. | |
| Denmark | 180 mg. | |
| Belgium | 180 mg. | |
| U.S.A. (most) | 150 mg. | |
| Victoria (Australia) | 230 mg. | |

Due to innate differences in individuals both as to the examining medical man and the potentially impaired driver, medical examination alone is considered inconclusive as to driving safety.

A practical approach to this problem would be to strike a level of say, 100 mg. per 100 ml. of blood, above which it would be an offence to operate a motor vehicle, irrespective of any other factors such as tolerance or habituation. The driver should retain the right to have an independent concomitant analysis by another analyst.

2) Speed, per se, is purely relative to any given accident situation and actually is a driver hazard. Excessive speed is a factor in many accidents but is not easily subject to proof. Recent more stringent controls in this field are producing results. Governors on cars would be no more infringement on personal liberty than laws against suicide. In New Providence Island, U-drives have an ingenious built-in answer to speed. The horn blows if the speed limit is exceeded, and there is a fine for tampering with it.

Many commercial vehicles are equipped with tachographs — a graph device which records time and all movements of the vehicle in terms of m.p.h. This has constituted prima facie evidence on occasion, and would, if used, reduce the number of high speed accidents.

3) Highway conditions are the subject of continual review by the Highways Branch of the Province in the light of each and every accident which occurs. Sealer coats on the newer highways make them extremely treacherous in the first ten to fifteen minutes of a rain storm, and more warning signs to this effect would minimize this type of accident.

White lines along the outer edge of the travelled portion of highways, as well as the centre, would make night driving safer and reduce the number of auto-pedestrian accidents. The number of fatalities at poorly protected railway crossings speaks for itself.

4) Mechanical defects in cars and trucks account for less than 3% of fatalities. Defects in structure and design increase both mortality and morbidity. Stylists claim that pride and excitement of ownership dictate the lines of an auto, rather than stability and safety.

In "rollovers," ejection dominates the picture, while in head-on collisions, ejection plays a minor part. Death chance in rollover for the ejectee is one in ten, while for the non-ejectee it is one in one hundred. How simple it seems to put an extra ¼ inch of steel on a bolt-action door lock to keep the door closed!

The Research Programme at Cornell University has made an intensive study of the use of seat belts and state their use to be "The most important single economically feasible device available to control trauma." An estimated 25% reduction in fatalities is surely worth striving for!

The objections to their use where fire or water is a factor, are greatly outweighed by the infrequency of such events. The co-operation of insurance companies to offer reduced premiums, if installed, should be sought.

Three recent accidents involved a car and gravel truck. The car, in each case, ricocheted off the front of the truck. The accident would have been little more than a side swipe, but for the protruding square box behind, which killed each of the drivers. A tapered box or rounded corners would have allowed the ricochet to continue!

Whip injuries to the neck are becoming increasingly common in rear end collisions. A simple head rest would eliminate this entirely!

Ornaments on hoods seem to be designed to impale pedestrians, and are quite unnecessary. Padded dash boards and elimination of instruments and knobs from their present location, would minimize injuries. With power steering, the conventional steering wheel is unnecessary.

Wide, square fronts, ornate grilles and flared fins of today's automobile are a far cry from the bullet-shaped configuration the physicists portray as offering least resistance.

Much of this data on safety has been developed and tested and the bottleneck is in the installation on vehicles. Co-operation of groups with adequate public support is necessary. If some of the enthusiasm shown in the preliminary investigation and subsequent litigation were diverted to include a survey of the immediate cause of physical injuries it would produce a battery of statistics which would have to be recognized. Such surveys have been done sporadically in the past, but a nation-wide survey, headed by the profession, is overdue.

**Summary**

Statistics for fatal and non-fatal accidents in Canada and Manitoba are quoted. The handling of accident victims on the road and emergency treatment in hospital is discussed.

Factors involved in causation of accident situations are reviewed in terms of driver, equipment and highways. Remedial measures are suggested.

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Statistics courtesy Motor Vehicle Branch, Province of Manitoba.

# Head Injuries

**Rankin K. Hay, F.R.C.S.(C)**

The seriousness of a head injury depends upon the degree and extent of the damage done to the brain, either at the time of the accident, or subsequently as a result of complications. The brain may be concussed, contused or lacerated. Later it may be compressed by an effusion of blood, or infected by organisms which gain access to it through an open wound. Fractures of the skull are of secondary importance for, if not compound or depressed, their only significance lies in the fact that they give some indication as to the force of the blow to the head. The diagnosis of a brain injury and the recognition of complications rests essentially on the clinical picture.

## The Pathology of Cerebral Trauma

It is of fundamental importance to distinguish between primary damage which is inflicted at the time of the injury and secondary damage which develops later as a result of complications.

### Primary Brain Damage

Three types of direct brain injury are described. In order of increasing severity these are: concussion, contusion and laceration. They may occur separately or in varying combinations. The clinical state of cerebral concussion requires description for the term is used much too loosely. It is characterized by immediate loss of consciousness, but with rapid recovery of the senses. If examined at once a concussed patient would be found to be in deep coma, pale, with shallow respirations and a slow feeble pulse; the limbs would be flaccid and all reflexes, even the corneal, pupillary, and swallowing reflexes would be unobtainable. Within a few minutes, however, such a patient would be fully conscious with all visceral and reflex activity re-established. The nature of the neuronal damage responsible for this state is unknown. Presumably it is the result of a transient derangement of intracellular function for no lesion, even of microscopic proportions, has ever been demonstrated in a case of pure concussion. We do not even know whether it is the result of a localized or a generalized paralysis of neuronal function. It is most easily produced by sudden acceleration as when the head is struck by a moving object, or by rapid deceleration such as occurs when the moving head strikes the floor. After such injuries it is the cerebral hemispheres which are most extensively damaged, however, it is known that the "central reticular formation" of the brain stem and hypothalamic region plays an important role in maintaining consciousness, and it is possible that neuronal paralysis in this structure is responsible for the transient coma. This may be a direct result of trauma to the neurones or it may be an indirect effect of transient ischaemia brought about by the vasospasm induced in the small basal perforating arterioles, for these are liable to be put on the stretch whenever the brain is suddenly moved within the cranium. The skull can be extensively fractured by a slowly applied compressive force, but, so long as this is not of overwhelming proportions, consciousness may be retained. Similarly in the case of penetrating wounds produced by high velocity missiles, most of the energy is expended in penetrating the skull and, provided no vital cerebral centre is damaged, there is no concussion.

In the case of contusion and laceration gross physical damage evident to the naked eye is done to brain tissue. Such lesions are most commonly seen in the vascular cerebral cortex but in more severe injuries may also be found in the subcortical white matter or even in the brain stem. Bleeding is an inevitable accompaniment and varies in degree from a few scattered cortical petechiae to massive intracerebral or subdural haemorrhage. If the patient survives, secondary oedema follows as a result of a local inflammatory reaction. Such lesions may be seen immediately beneath the site of impact when the head is struck by a blunt object or adjacent to a depressed fracture or in the depths of a penetrating wound. Owing to the way in which the brain can move and even swirl about inside the cranium, these destructive lesions are also found at sites remote from the point of impact. These latter are referred to as contra-coup injuries and may be seen over the medial aspect of a hemisphere which has been forced against the rigid dural partition formed by the falx cerebri, over the tips of the temporal lobes which tend to be torn by the sharp lesser wings of the sphenoid bone, or over the inferior surface of the frontal lobes if they have been violently moved over the irregular bony ridges on the orbital plates. Contusion may result also from a suction effect, as when the cortex is drawn away from its normal supporting wall. The nature of the abnormal neurological signs and symptoms which follow cerebral contusions will depend upon their site; intellectual impairment and personality changes result from widespread bifrontal contusions, hemiplegia, dysphasia, or homonymous hemianopias follow lesions in appropriate cortical areas, while prolonged coma and states of decerebrate rigidity accompany contusions of the brain stem.

**Secondary Events or "Epiphenomena"**

After cerebral contusion there is some secondary swelling of the neighbouring brain as a result of oedema. This, however, remains localized to the areas adjacent to the structural damage and generalized oedema is not common. Fatal cerebral compression is usually due to accumulations of blood. Haematomas may collect in the epidural plane, the subdural space, or within the brain substance itself.

Epidural haemorrhage most commonly occurs from a torn middle meningeal artery and is usually, but by no means always, accompanied by a fracture of the skull in the temporal region. Such arterial bleeding is brisk and a large haematoma may accumulate within a few hours. Bleeding from diploic channels, or from dural venous sinuses will lead to a more gradual accumulation of blood in the extra-dural plane. As a rule such haematomas produce clinical effects within twenty-four hours of the injury and they constitute one of the few really acute surgical emergencies.

Large subdural collections take longer to form. Acute ones are usually associated with extensive under-lying lacerations of the brain and tearing of cortical vessels. Subacute collections usually remain fluid and are the result of the tearing of "bridging veins" as they pass from the cortex to one of the large dural venous sinuses. A small subdural collection, insufficient to produce signs or symptoms, may become organized and a semi-permeable membrane then develops around it. Possibly as a result of osmosis a gradual increase in size of the haematoma occurs and this leads to delayed cerebral compression weeks or months later by what is then a chronic subdural haematoma. Such collections may be bilateral.

Post-traumatic intracerebral haematomas of large size are not common. They may be acute, a result of the immediate rupture of an artery, or they may be delayed when rupture occurs at a site in a vessel wall which was only weakened at the time of the injury.

Intracranial infections, meningitis, encephalitis, or cerebral abscess may all result in acute focal or generalized cerebral swelling and in compression which may be rapidly fatal.

**Fractures of the Skull**

Fractures may be simple linear cracks resulting from distortion of the skull, or they may be depressed or comminuted. As with fractures elsewhere in the body they may be closed or open. Compound fractures of the skull may communicate with the exterior either directly through a scalp wound — "external compounding," or indirectly through one of the paranasal air sinuses or the cavity of the middle ear—"internal compounding." After a compound fracture it is of vital importance to determine whether or not the dura mater has been torn. The escape of brain tissue or of cerebro-spinal fluid from a scalp wound, the external auditory meatus, the nose or the nasopharynx clearly indicates a breach in the continuity of the dura. In such cases air may be demonstrated within the cranium on x-ray examination. The risk of secondary intracranial infection in these cases is great.

In adults depressed fractures are usually accompanied by an overlying scalp wound. In children a large depression may be associated with an intact scalp. On the other hand the dura in these small patients is very firmly adherent to the bone and is more likely to be torn beneath the site of a depressed fracture. Cerebro-spinal fluid may collect beneath the intact scalp in such cases. Though a depressed fracture may compress the brain locally, it is rarely a cause of increased intracranial pressure. If clinical evidence of cerebral compression accompanies such a fracture it is almost invariably the result of an associated haematoma.

In cases of penetrating wounds the bone beneath the wound of entry is shattered and small fragments of bone are carried into the brain substance.

### Associated Injuries

Automobile accidents are responsible for the majority of head injuries in this province, and the increasing speed of vehicles is resulting not only in more serious intracranial damage, but also more frequently in major injuries to other parts of the body. A combination of chest and head injury is particularly dangerous. Major limb or pelvic fractures are potent causes of haemorrhagic shock. Injuries to the cervical spine are easily overlooked in a comatose patient. It is important to remember that a cervical fracture-dislocation may be present, even in a conscious patient, without evidence of injury to the spinal cord or the cervical nerve roots.

Facio-maxillary fractures may result in the displacement of an eye with resulting diplopia as well as damage to the peripheral branches of the trigeminal nerve. Cranial nerve palsies frequently complicate fractures involving the base of the skull or the bony orbit. The olfactory bulbs are often damaged and permanent anosmia ensues. Injury to an optic nerve or the chiasm likewise causes permanent impairment of vision. On the other hand the prognosis in post-traumatic paralysis of the 3rd, 4th and 6th nerves is good, provided the peripheral nerve only has been involved. Ocular palsies due to involvement of the nuclei may be seen in severe injuries with brain stem contusion and are then a grave prognostic sign.

The facial nerve occasionally is injured directly and immediately after a fracture involving the petrous bone. More commonly facial paralysis is a delayed event and results from the secondary compression of the nerve within the facial canal by haemorrhage or inflammatory swelling which develops around such a fracture. The prognosis in these "early delayed" types of post-traumatic facial palsy is favourable. The auditory and vestibular nerves are rarely damaged directly; post-traumatic deafness, vertigo, or unsteadiness may, however, result from injury to the appropriate end organ. Injury to the last three cranial nerves is very unusual.

### Diagnosis

The circumstances of the accident, and the subsequent rapid recovery of the patient may make the diagnosis of trauma self evident. This, however, is not always the case when coma is prolonged, for the patient may have collapsed as a result of some systemic disease such as epilepsy, spontaneous intracranial haemorrhage, diabetes, alcoholism or uraemia, and the head injury is then only a secondary event. The past history and particularly the history of events immediately preceding the accident are therefore of great importance. If the patient is unable to provide a clinical history, this must always be sought from relatives, friends or witnesses.

Loss of consciousness is the most important single physical sign after head injury. Coma always accompanies cerebral concussion, and though it may be short lived, it is invariably followed by amnesia for events immediately preceding and following the blow. A history of such amnesia is very strong presumptive evidence that cerebral concussion has been present. Contusion and laceration are associated with a more prolonged period of unconsciousness and with a more gradual recovery. Indeed the length of coma is a reliable guide to the severity of the cerebral injury. During his recovery a comatose patient will pass through a period of semi-coma when he will respond purposefully only to painful stimuli, and later through stages of restlessness and mental confusion before the senses are finally and completely restored. Confused and violent behaviour may be regarded as a result of the absence of those inhibitions which normally emanate from highly developed and specialized cortical neurones. It is, however, often aggravated by pain and discomfort, and causes of irritability such as headache, or the discomfort of a full bladder should be looked for. Meningeal signs may be prominent. Traumatic subarachnoid haemorrhage leads to a varying degree of headache, neck stiffness, fever, vomiting and photophobia. In the more severe cases when coma is profound numerous autonomic disturbances are seen. Tachycardia, hyperthermia, overbreathing, hypersecretion of mucus from bronchi, vomiting, gastrointestinal stasis and haemorrhage. All these demand urgent attention. They are the result of direct physical damage to or secondary ischaemia of the autonomic regulating centres in the hypothalamus.

Everyone who has sustained a head injury must be kept under observation, for we have no way of knowing which patients are going to develop complications. Secondary cerebral compression as a result of intracranial haemorrhage can follow the most minor accident. In a patient who has never been unconscious, or in one who has recovered his senses after concussion it is fairly easy to determine the onset of cerebral compression, for this is heralded by severe headache, gradual and progressive drowsiness, coma, and the appearance of focal neurological signs. The onset of this serious complication is not so easily detected in a patient who has been in coma ever since the accident, but any deterioration in his level of consciousness or the appearance of other clinical signs indicative of the formation of a tentorial pressure cone must none the less be carefully watched for. In such cases, accompanying the deterioration in the level of consciousness there will be certain neurological signs of a paralytic order. The pupil on one side, usually the side of the intracranial haemorrhage, becomes widely dilated and loses its normal reaction to light. Later the other pupil follows suit. The contralateral limbs become paralyzed and their tone abnormal, sometimes flaccid, sometimes spastic; the tendon reflexes are correspondingly lost or

increased. The plantar responses, if obtained, become extensor. If intracranial tension is not relieved the limbs on both sides become paralyzed and the patient may then assume a posture characteristic of decerebrate rigidity with bilateral fixed and dilated pupils and bilateral extensor plantar responses. Respirations become deep, laboured, and noisy and a Cheyne-Stokes rhythm develops; pulmonary secretions increase and accumulate and the patient's face becomes congested. Occasionally an increase in the blood pressure, and a bradycardia are seen in the early stages of mounting intracranial pressure; more commonly, however, this is not observed and the pulse rate steadily rises while the blood pressure falls. The crux of the matter lies in repeated observation and in the recording of the level of consciousness and the vital signs in order that any change in the initial "basic state" may be detected at the earliest possible moment.

**The Management of Closed Uncomplicated Head Injuries**

Intracranial bleeding is never copious enough to produce oligaemia. The presence of surgical shock in head injured patients is either an indication of serious blood loss from the scalp or, if this has not occurred, of severe bleeding elsewhere in the body. The airway requires immediate and urgent attention in all comatose patients, for it is liable to be obstructed by vomit or by the falling back of a paralyzed jaw and tongue. In addition excessive bronchial secretions rapidly obstruct the smaller bronchi and bronchioles. Comatose patients should be nursed in the lateral or in the semiprone position at all times, and they must be turned from side to side every two hours (Fig. 1.) In this position the airway can be kept clear for pulmonary secretions will gravitate from the uppermost lung along the trachea and pool in the dependent cheek. The mouth and pharynx should be regularly cleaned with moist gauze or by suction. When the patient is turned onto the opposite side the other lung will likewise be drained. When secretions are copious, the foot of the bed should be raised so as to take more advantage of the force of gravity. Never nurse unconscious patients on their backs nor with the head elevated. Oxygen can with advantage be administered, for anoxia will cause more cerebral damage. Such measures are usually adequate but if respiratory embarrassment continues or recurs there should be no hesitation in resorting to bronchoscopy or tracheotomy. It must be remembered, however, that even after these procedures meticulous nursing care is still essential. Prophylactic antibiotics may be used to prevent acute respiratory infection.

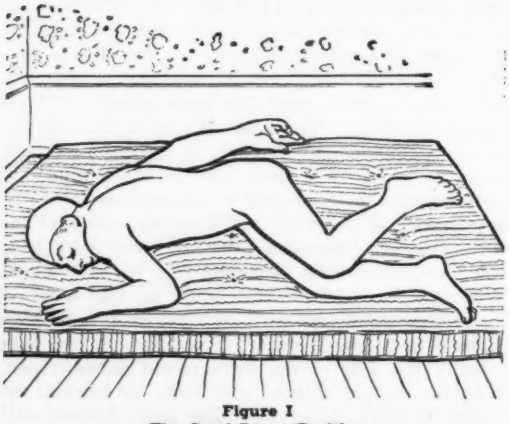

**Figure I**
**The Semi Prone Position**

In this position the airway can be kept clear and the risks of inhaling vomit or blood are minimized. Note that the patient is lying on a mattress placed on the floor; he cannot fall and thus requires no physical restraint.

Pyrexia, over 102° to 103° F., and over-breathing must be combated by cooling the patient. This is best accomplished by simple measures. Remove the bed clothes and keep the room cool. If this does not suffice, then cover the patient with a damp sheet and blow a current of air over it with a fan. If shivering is thereby produced it should be counteracted by small doses of chlorpromazine, 25 gms. given by intramuscular injection and repeated as required. This technique should be persisted until the rectal temperature is normal.

During the first day special attention to the administration of fluids is unnecessary. Subsequently tap water may be given per rectum or intravenous fluids may be commenced. In a restless patient such routes may prove impracticable, and in any case after a day or so adequate nourishment cannot be given by either of these methods. On the second day an oesophagus tube of the Levin or Jacque's type should be passed, and, once it has been established that fluid given via the tube is not accumulating in the stomach, then regular feeding can begin. Suitable tube feeding diets are listed in the appendix at the end of this paper. Great care must be taken to ensure that the end of the tube is actually in the stomach when feeding is in progress. Nothing must be administered by mouth until it is quite certain that the sucking and swallowing reflexes have returned. After this teaspoonfuls of water can be given, and later a feeding cup can be employed. Under no circumstances must fluid be forced into the patient's mouth by a syringe.

Proper attention must be given to the skin, especially over the various bony pressure points and in the perineum. The patient should be turned at least every two hours and the skin must be kept clean and dry. The care of the bowel and bladder present special problems. The bowels are usually constipated for the first few days, subsequently they will evacuate spontaneously or after an enema. The bladder empties reflexly from the beginning, retention of urine is rarely seen. These patients should not be catheterized, for this rapidly leads to a urinary tract infection and furthermore in a semicomatose or confused patient a catheter is a potent cause of irritation and restlessness.

Once the patient is conscious he may take up any position in the bed he wishes. There are no hard and fast rules, and, as his symptoms subside, he may be given increasing liberty and sit up for meals. How long should he remain in bed? The best criterion is freedom from headache, and once this has been settled for a few days he can be allowed up to the bathroom and given gradually increa. ing freedom. When physically active, graduated exercises should be prescribed. Residual limb paresis, diplopia, or dysphasia will require special advice.

**The Place of Radiography and Lumbar Puncture**

X-ray examination of the skull is unnecessary in the early stages of management of closed head injuries, for it does not influence the initial treatment. There are dangers attendant on such an examination in a comatose patient. There may be delay in establishing a clear airway, the necessary manipulations of the head may further obstruct the airway or there may be an unrecognized injury to the cervical spine. Furthermore radiographs done as an emergency measure, on a patient who is restless, unco-operative and probably vomiting, are unlikely to be satisfactory. If evidence of cerebral compression develops within the first 24 hours, this is probably due to an extradural haematoma and the proper place for such a patient is the operating room. Valuable time may be lost if attempts are made to get preliminary radiographs of the skull.

Lumbar puncture may be of great value, but it is potentially dangerous. It should never be done if there is any indication that acute cerebral compression is developing. There is very rarely any indication for it within the first 24 hours; after this, however, it may provide information of considerable diagnostic value and may prove beneficial therapeutically. When it is employed the pressure should always be recorded with the patient lying relaxed in the left lateral position. The drainage of C.S.F. will lower intracranial tension and may relieve headache. Some authorities consider that the repeated drainage of C.S.F. during the acute and convalescent phases of the illness helps to prevent the formation of subdural meningocerebral adhesions which may be a cause of post-traumatic headache. It is a necessary diagnostic measure if meningitis is suspected. As a general rule it is best to do a lumbar puncture in head injury cases only on the recommendation of the surgeon who would be called upon to operate should this become necessary.

**Surgical Treatment and the Question of Evacuation**

It would be inappropriate here to discuss the various surgical operations applicable to cases of cranio-cerebral trauma. Each case must be judged individually, and the general practitioner should decide after his initial assessment whether he can deal with the local surgical problem and the wider problem of general management himself or whether the patient should be evacuated to a neurosurgical centre. Many factors will influence such a decision, the severity of the local trauma, the probability of complications, the facilities for nursing, anaesthesia and surgery which are locally available, and most important, the accessibility of specialist help. Uncomplicated head injury patients travel well provided there is no serious associated chest, abdominal, or limb injury and provided oligaemic shock and respiratory obstruction are combated. The only complication in which time becomes of extreme importance is the acute epidural haematoma. These comprise about 3% of all civilian head injuries. A doctor who is in a remote country district should, therefore, be prepared to do exploratory cranial burr holes and, on locating an extradural haematoma, a simple craniectomy in order to provide a cerebral decompression. Problems arising in the management of acute cerebral compression, or in making decisions for or against transportation can often be profitably discussed over the telephone with a neurosurgical consultant who would be only too willing to give practical advice and help. In general terms it may be stated that those patients who require transfer to a special centre include the following types of case:

1) Those in whom a definite "latent interval" of consciousness has occurred.

2) When progressive drowsiness develops in a patient who was alert or no more than confused at the time of the accident.

3) Those who become deeply comatose and develop a fixed and dilated pupil.

4. When there are fractures involving the paranasal air sinuses or the middle ear, with or without the escape of C.S.F. or the presence of intracranial air.

5) Those with wounds over the calvarium when the dura mater has been penetrated and when brain tissue or C.S.F. are seen escaping from the wound.

6) Compound, depressed or comminuted fractures with evidence of underlying cerebral damage, or laceration of dura.

As for the milder cases of concussion, uncomplicated scalp laceration and skull fracture; any doctor is competent to deal with these provided he is on the watch for complications, can keep the patient under constant observation, provide adequate nursing care, and so long as he is willing to guide his patient through the periods of convalescence and rehabilitation which may prove long and troublesome. The scalp should be carefully shaved and washed for a distance of at least three inches around small lacerations. Even the smallest wound should be carefully explored, visually and with the finger, for scalp wounds are notoriously deceptive and a minor abrasion or laceration may hide an extensive injury to the underlying muscle, bone or dura. All debris should be removed. Simple linear cracks in the bone may be left undisturbed if they are not obviously contaminated. The wound should be dusted with penicillin and sulphonamide powder

or with bacitracin and sutured with interrupted stitches of fine silk. If in the course of such an exploration a depressed fracture is encountered, or if a dural tear is seen it is quite legitimate simply to close the wound with a few sutures or to pack it lightly with sterile gauze and transfer the patient to a bigger centre. Whenever possible all scalp surgery should be done under local infiltration anaesthesia.

Bleeding from the nose and ear present special problems. The nose should never be plugged, the blood is thereby simply diverted into the nasopharynx and the risks of aspiration and infection increased. Bleeding from the ear should be dealt with by covering the pinna with a large pad which is bandaged into place. Never plug the ear canal, and never attempt to clean it out. Always watch for C.S.F. leakage in these cases, if this occurs, it will probably become evident on or after the second day when the bleeding has subsided.

Associated facial fractures even with displacement may be masked by the accompanying swelling, and if these are left untouched for more than a few days it becomes exceedingly difficult to manipulate them back into position. Early consultation with a facio-maxillary surgeon is recommended. A displaced maxilla may lead to a displaced eye and diplopia.

### The Sequelae of Head Injury

The terms "post concussional syndrome," or "post traumatic syndrome" which have become common usage should be abandoned for they are vague and meaningless and patients with a variety of quite unrelated complaints are grouped together under such labels to the confusion of everyone. Many symptoms of a minor nature are encountered after head injury. Headache, "dizziness," true vertigo, memory impairment and inability to concentrate—all occur and it is often very difficult to determine to what extent these are initiated by organic damage, by natural or pathological anxiety or by the influence of impending litigation.

**Post Traumatic Headache**

If due to physical causes, this must arise from pain sensitive structures in the head and can therefore only originate from the scalp, certain areas of the dura mater or the basal cerebral arteries. These structures will become more sensitive when acutely inflamed as after acute subdural haemorrhage or pyogenic meningitis. Headache may be associated with expanding intracranial lesions which distort the brain and the dural partitions and stretch the basal cerebral arteries and arterioles. Common causes are chronic intracranial haematomas and abscesses be these extradural, subdural, or intracerebral. Post traumatic headache may result from tension on fine subdural adhesions which are allegedly formed over swollen and contused cortex. It is said that such headache is less likely to occur when there has been a spontaneous leakage of C.S.F. or if repeated lumbar punctures have been done.

**Post Traumatic Vertigo and "Dizziness"**

The term "dizziness" is used very loosely and usually refers to sensations which the patient cannot describe clearly. An effort should always be made to determine exactly what a patient means by this word; much confusion will then be avoided. The term vertigo should never be employed unless there is a subjective sensation of motion either of the patient or of his surroundings. Real vertigo is usually paroxysmal, often initiated by movement or the assumption of a particular posture, it is always accompanied by nystagmus and often by deafness.

**Post Traumatic Neurosis**

Injuries to the head have serious implications and grave significance in the minds of most people, particularly is this so in the case of relatives and friends. It is of the greatest importance therefore that the very natural anxiety which both the patient and his relatives may have as to the eventual outcome of the injury be dealt with in an understanding and common-sense manner from the earliest stages of the illness. Some patients need repeated reassurance in order to prevent them from believing that their brain has been seriously damaged. Provided an adequate initial period of rest is ensured, provided physical activities during convalescence have been suitably graduated, and provided undue anxiety and worry have been allayed, patients should be encouraged to ignore their purely subjective symptoms and persevere with their rehabilitation. With patient, tactful but firm handling their self confidence will return. Always make a careful assessment of the type of work to which a man is returning and of his ability to do the work. A doctor's letter recommending that employers allow shortened hours for the first week at work helps in the rehabilitation process.

**Post Traumatic Brain Damage**

If considerable brain damage has occurred residual sequelae may be permanent. Intellectual deterioration with impairment of memory and inability to concentrate may follow the generalized and widespread damage produced by a prolonged period of increased intracranial pressure. In such circumstances treatment can only be prophylactic. In the early stages repeated lumbar punctures or a cerebral decompression may be needed to keep the intracranial tension within normal limits. A space occupying lesion such as a haematoma, a cyst, or an abscess may be responsible. Focal neurological signs such as hemiplegia, hemianopia and dysphasia are of course due to damage to the appropriate brain areas. Residual cranial nerve palsies are seen, but, except in the case of the olfactory and the optic nerves, there is a strong tendency to recovery. Anosmia is common after injury to the frontal lobes and may therefore be associated with some intellectual impairment.

**Post Traumatic Epilepsy**

Epilepsy may develop early or late after head injury, the incidence reaches its highest peak about the end of the first year. The fits may be localized (Jacksonian seizures) or generalized. Epilepsy is more common after cortical contusion or laceration and much more common after open wounds in which the dura mater has been torn than after closed injuries. After penetrating or infected brain wounds there may be extensive cerebral scarring, and the incidence of post traumatic epilepsy in such cases is 50%, whereas it follows only about 5% of closed injuries. No reliable prognostications are possible as to which patient will and which will not develop seizures. In order to diminish the risks it is advisable to prescribe anticonvulsant medication whenever there has been penetration of the dura. Only a small dose, Phenobarbitone gr. ½ B.D., may be needed but it should be taken for a period of at least three years after the injury.

**Post Traumatic Infection**

An intracranial infection is inevitable if a defect in the dura mater is allowed to persist in cases both of "external" and of "internal" compound fracture. Though small dural tears may heal spontaneously this is too uncertain a process to be left to chance and in such cases the dura should be repaired surgically. In the past cerebro-spinal fluid rhinorrhea or otorrhea was treated medically and operation was reserved for those cases in which the leakage persisted or recurred. This conservative approach was due to the fact that the leakage usually stopped after a few days. When this occurred it was considered that the dural defect had been closed by natural processes. Further experience however, has shown that many of these patients, in spite of such evidence of healing, develop a delayed intracranial infection with or without a recurrent fluid leak, and many of them die. In fatal cases it is usually found that the dural defect has in fact not healed, and that a free communication exists between a paranasal air sinus or the middle ear and the subarachnoid space. The initial lack or the early cessation of C.S.F. leakage may be due to the fact that the dural defect becomes temporarily sealed either by arachnoidal adhesions or by a small hernia of brain tissue. Today immediate intradural exploration of a fracture over the cribriform plate, the tegmen tympani or the mastoid air cells is advocated by some even when a dural defect in dura can be readily repaired by covering them with a graft of fascia.

**APPENDIX**

**Tube Feeding For Comatose Patients**

1. **First 24 hours**
   No fluid by mouth.
   No intravenous infusions.
   (Blood Transfusion only when indicated).
2. **Second 24 hours**
   (a) Pass oesophagus tube by mouth. If there is a fracture of skull base a Levin type tube should be passed via the mouth, not through the nose.
   (b) 2 oz. water given every 2 hours for 6-8 hours. If this does not accumulate in stomach — as proved by failure to aspirate all fluids at end of 6-8 hour period, proceed to "c".
   (c) Milk, glucose and water Regeme.
   3 pints whole milk
   3 pints water
   300 grams glucose.
   Above mixture prepared and given in divided doses over 24 hours.
3. **Later if patient** progresses satisfactorily a High Protein Diet No. 1 or No. 2 is substituted on 4th or 5th day.

| | Diet No. 1 | Diet No. 2 |
|---|---|---|
| Whole Milk | 40 oz. | 40 oz. |
| Eggs | 2 oz. | 4 oz. |
| Full Cream dried milk | 3 oz. | 6 oz. |
| Soya flour | 2 oz. | 2 oz. |
| Glucose | 4 oz. | 4 oz. |
| Salt | 5 gms. | 5 gms. |
| Ferrous Sulphate | 5 grains | 5 grains |
| Aneurin Hcl. | 5 mgs. | 5 mgs. |
| Riboflavin | 2 mgs. | 2 mgs. |
| Nicotinamide | 20 mgs. | 20 mgs. |
| Pyridoxine | 2 mgs. | 2 mgs. |
| Ca. Pantothenate | 3 mgs. | 3 mgs. |
| Ascorbic Acid | 200 mgs. | 200 mgs. |
| Vit. A. | 4500 int. units | 4500 int. units |
| Vit. B. | 1000 int. units | 1000 int. units |

| Diet No. 1 Provides | Diet No. 2 Provides |
|---|---|
| 225 grams Carbohydrate | 250 grams Carbohydrate |
| 100 grams Protein | 130 grams Protein |
| 100 grams Fat | 130 grams Fat |
| 2200 Calories | 2700 Calories |

**Instructions for use:**

1. Both diets are diluted with water so that a Total Volume of six pints is given every 24 hours.
2. If diarrhoea develops, glucose content is reduced or removed.

. . . "I remember an epidemic disease, of a very dangerous and fatal nature, raging in the towns around me a few years ago. When the storm, which had swept away an endless number of people had passed over, one of the most reputed physicians of the whole district published a pamphlet on the subject, in which he changed his mind about blood-lettings which had been practised, and confessed that that was one of the chief causes of the mortality that ensued. Moreover, the writers of the faculty hold that there is no medicine that does not contain some harmful ingredients; hence, if those which benefit us also do us some harm, what must those do that are totally misapplied?"

Montaigne - Essays.

# The Crushed Chest

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The severely crushed chest is probably one of the most devastating of injuries. It disrupts the respiratory, cardio-vascular, cerebral and genito-urinary systems. It accounts for 25% of reported deaths in car accidents. It is accompanied by other serious injuries in 50% of cases. Its treatment takes priority over all other injuries.

The early diagnosis of a crushed chest is often missed. The mobile segment seldom is seen to flail wildly with respiration. The patient is not often deeply cyanosed. However, he is suffering from acute respiratory failure. Hypercarbia is the basic defect, not hypoxia. Therefore, the administration of oxygen alone may be of little or no value. Careful inspection of chest wall will demonstrate paradoxical movement of the injured side with each phase of respiration. However, extensive hematoma may obscure this. Routine palpation of the chest wall in all multiple injury patients should be performed.


**Figure 1**

Figure 1 demonstrates how this is done. Paradoxical motion can be easily and most accurately detected by this method. Relatively slight paradoxical movement over a large area of the chest can produce profound respiratory distress. Initially the paradox may be minimal because of the interdigitation of the fracture lines. But with the continuous movement of respiration these break down and the degree of paradox increases greatly.

The fundamental problem of a crushed chest is acute respiratory failure. This is the result of:

1) alveolar hypoventilation
2) chest wall instability

In normal respiration the negative intra-thoracic and intra-pleural pressures are equal in each chest cavity. Thus air is drawn into both lungs from the upper respiratory tract in approximately equal volumes. The converse is true of expiration. 150-175 cc. of this air does not take part in the gaseous exchange of oxygen and carbon dioxide in the alveoli. This volume represents the dead air space — the amount of air required to fill the upper respiratory tract, trachea, and bronchi to the end of the terminal bronchioles.

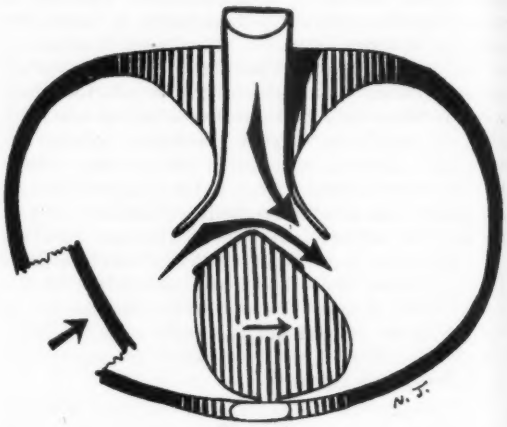

**Figure 2**
**Inspiration**

In a patient with a crushed chest a similar negative intra-thoracic and intra-pleural pressure is created on inspection. But the positive atmospheric pressure collapses the mobilized segment of the chest wall (Fig. 2). This results in a smaller negative pressure or lesser vacuum being created on the injured side. A pressure gradient is created within the chest itself. As a result, on inspiration varying amounts of air are drawn from the injured lung into the sound lung as well as from the upper respiratory tract. On expiration as Fig. 3 illustrates the converse is true.

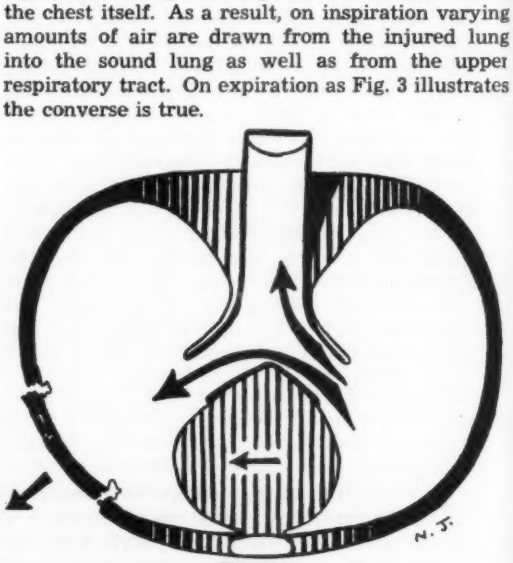

**Figure 3**
**Expiration**

Again a pressure gradient results, as much of the positive pressure on the injured side is expended on shifting the flail segment. And thus alveolar air already heavily contaminated with $CO_2$ is transferred from the sound lung to the injured lung.

This phenomenon is known as "penduluft," paradoxical respiration, bucketing or rebreathing of air. It at once becomes obvious that the CO2 tension of alveolar air will be very high. Therefore the partial pressure of carbon dioxide (PCO2) of the blood will be well above normal and a corresponding drop in blood PH will occur. This produces respiratory acidosis. The respiratory centre is sensitive to changes of PCO2 and PH and relatively insensitive to changes of PO2. It is therefore stimulated initially in respiratory acidosis in an attempt to drive off the excess PCO2. However, because of the altered dynamics of respiration described above this cannot be acomplished. Rather hyperpnoea makes the situation worse by increasing rebreathing and a vicious cycle is established which, if permitted to continue, will end in death. In addition respiration may be further impaired by hemopneumothorax or by accumulation of secretions and atelectasis with subsequent pneumonia. Not only is air rebreathed, but also purulent secretions, blood clot and casts are coughed ineffectually from one lung to the other. The usual clinical signs of atelectasis are altered because of the altered air flow. They are unreliable. Thus complications arise unnoticed until it is too late to effectively correct them.

Further physiological upset occurs as a result of altered respiratory dynamics. Venous return to the heart is impaired and low output failure may result. A high arterial PCO2 causes peripheral vasodilatation producing shock. This is in addition to that created by blood loss of the injury itself and those that may accompany it. This depresses renal and cerebral function. As the biochemical and clinical state of the patient deteriorates so these secondary factors are magnified. Two aims must be satisfied if therapy is to be successful.

1) adequate alveolar ventilation must be provided,
2) chest wall stability must be secured.

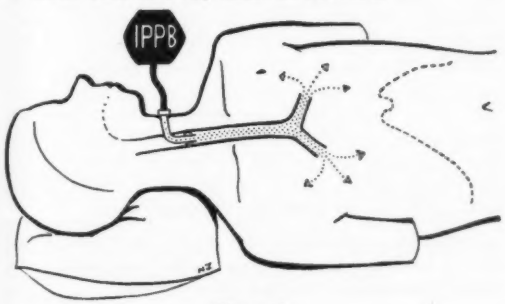


Figure 4

Both can be achieved with continuous intermittent positive pressure breathing (Figure 4). Air delivered to the tracheo-bronchial tree under positive pressure eradicates "penduluft" or rebreathing. It, therefore, quickly corrects respiratory acidosis by blowing off CO2. It also provides a positive pressure air cushion for the chest to ride on symmetrically giving linear rib stability without interfering with rib mobility. The pressure can be regulated to promote venous return and avoid low-output failure.

An apnoeic state is created by overbreathing which produces mild alkalosis to depress the respiratory centre. In this way complete control of respiration is obtained.

**Management**

1) Initial Acute Phase
2) Chronic Phase

**Management of Acute Phase**

1) Establish a Clear Airway
   a) endotracheal intubation and suction
   b) bronchoscopy
   c) tracheotomy with humidifier.
2) Establish Continuous Intermittent Positive Pressure Breathing—preferably through a cuffed tracheotomy tube.
3) Evacuate Pleural Cavities with underwater tube drainage.
4) Replace Blood Loss.
5) Intubate Stomach.
6) Avoid Sedation.

In comment on Table 1 a tracheotomy achieves the following:

i. reduces the anatomical dead space by 50-75 cc.
ii. reduces turbulence and resistance to air flow.
iii. facilitates tracheo-bronchial toilet by catheter suction or bronchoscopy through the tracheotomy.

Adequate humidity must be provided to prevent drying of mucus and bronchial cast formation. The effect of tracheotomy is dramatic. In the absence of acidosis it may be unnecessary to use continuous positive pressure breathing.

In evacuating the pleural cavities a large intercostal tube should be used. Positive pressure breathing or tube suction is necessary to obtain lung expansion because of altered intrapleural dynamics.

**Management Phase 2**

i. daily bronchoscopy and at least half hour tracheotomy suction.
ii. overbreathe patient maintaining alkalosis.
iii. provide adequate antibiotic prophylaxis.
iv. maintain electrolyte balance (hypopotassemia).
v. provide adequate caloric intake by tube feeding.

The chronic phase is distinct, and has its own peculiar problems. The airway must be kept clear. Purulent secretions, bronchial casts and blood clots must be frequently removed. The problems of infection and atelectasis in this phase of management are particularly significant. It reaches its greatest magnitude when adequate initial therapy has not been provided and the patient is first seen on the third day when the chest becomes fully mobilized and infection is established. The gastric tube is

initially used to decompress the stomach but when a positive gastric balance is established it can be used for tube feedings. The tremendous expenditure of energy in these injuries requires careful provision for the metabolic need. This chronic and convalescent phase will extend from one to four weeks depending on the severity of injury and the rate of rib healing.

**Complications**

1) ruptured diaphragm, liver, spleen, kidney.
2) cardiac contusion, delayed or early rupture or aneurysm of aorta, cardiac tamponade intercostal artery hemorrhage.
3) fat embolism.
4) venous embolism.
5) other injuries.

The complications and other injuries in association with a crushed chest injury must be treated early and expeditiously. The risks are great, but the risk of watchful waiting is greater. Proper timing and proper choice of procedure is mandatory.

**Summary**

The physiological basis for the management of a severely crushed chest has been outlined. The fundamentals of management briefly discussed. The emphasis has been laid on early complete comprehensive management and constant surveillance.

## Trauma of the Heart and Thoracic Aorta

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Wounds of the heart and aorta may result from penetrating or non-penetrating trauma. Penetrating injuries may be due to stab wounds, commonly knives and ice picks, or missiles. Non-penetrating heart and aortic wounds are usually associated with thoracic cage trauma such as the steering-wheel injury. Cardiac injuries may result from compression of the abdomen or minor direct trauma such as a blow from a fist.

**Penetrating Cardiac Injuries**

The first attempt at repair of a stab wound in the human heart was by Cappelen[1], in 1895 but this was unsuccessful. In 1896 Rehn[2], of Frankfurt, Germany performed the first successful repair of a stab wound of the heart. According to Vaughan[3], Launay, in 1902 performed the first successful repair of a gunshot wound of the heart. By 1909 Peck[4], was able to report on the operative repair of 160 cardiac wounds with a mortality of 64%. Since then the procedure has become common.

Penetrating wounds of the heart are not uncommon. Parmley[5], has stated that they occur in 2-3% of all penetrating wounds of the thoracic cage. In civilian practice the offending weapon is usually a knife or ice pick. In military practice it is more commonly a missile.

**Classification of Penetrating Wounds of the Heart (According to Blau[6])**

Group I — Gunshot or other large penetrating wounds which produce severe injury.

Group II — Lacerated epicardium only or a lacerated epicardium plus an incomplete laceration of the myocardium.

Group III — Entry of the weapon into the heart chambers or laceration of a coronary vessel.

Group IV — Pericardiopleural communication.

**Pathology**

Cases included in Group I are usually instantly fatal and of no clinical importance.

Groups II and III are of greatest clinical significance for it is within this group that most salvage may be obtained. The cardiac injury per se is of minor importance. The accompanying hemopericardium and consequent cardiac tamponade, however, may be fatal, if not treated urgently and adequately. As bleeding into the pericardial sac occurs, the intrapericardial pressure rises and exerts pressure on the heart. The filling of the venae cavae and atria is reduced and the cardiac output is reduced. The venous pressure is consequently raised, the arterial pressure falls and the heart action is diminished. The normal venous pressure of 10 cm. of saline may be raised to 25 - 30 cm. of saline. A rise above 40 cm. is usually lethal. The site of bleeding may be epicardial vessels, one of the heart chambers or the intrapericardial aorta or pulmonary artery. Bleeding may temporarily cease due to tension in the pericardial sac or thrombus formation. Bleeding from the atria is usually more persistent than from the ventricles because of the thin muscular walls and their lesser ability to contract. Although the pericardium will also be lacerated, it is frequently not sufficient to allow decompression and the opening may be sealed by clot thus allowing tamponade to occur. Beck[7], states that rapid accumulation of 300 cc. of blood in the pericardial sac is sufficient to cause death.

Group IV is characterized by hemorrhagic shock due to internal and external blood loss which demands immediate surgical intervention.

**Diagnosis**

Any penetrating injury of the chest, upper abdomen, or neck is a potential cardiac injury. Associated pulmonary injuries such as tension pneumothorax, or hemothorax must be recognized and treated on their own merits. Persistent unexplained shock with cyanosis or air hunger should make one suspect cardiac tamponade. Beck's[8] triad

of: 1) falling arterial pressure, 2) rising venous pressure and 3) a quiet heart is indicative of cardiac tamponade. Fluoroscopy will confirm the reduced cardiac action and may be satisfactorily performed in the supine position.

**Management**

In the absence of cardiac tamponade or excessive hemorrhage a conservative approach with close observation is warranted. Antibiotics and blood replacement should be instituted. Occasionally the bleeding may cease temporarily due to thrombus formation, only to recur.

In the presence of cardiac tamponade, pericardiocentesis should be performed without delay and the operating room prepared for an emergency thoracotomy.

**Procedure of Pericardiocentesis**

With the patient supine a No. 17 short bevelled needle is inserted at the junction of the xyphoid and left costal margin. It is directed superiorly and posteriorly with suction on the syringe. Entering the pericardial sac imparts a sensation similar to that felt during a lumbar puncture. Dark fluid blood will be encountered. If successful there will be dramatic improvement in the patient. Failure may be due to clotted blood in the pericardial sac or a rapid recurrence of bleeding.

After successful paracentesis the patient must be observed closely for recurrence of the tamponade.

Emergency thoracotomy will be indicated if a) the paracentesis is unsuccessful, b) there is a recurrence of tamponade or c) there is pleuropericardial communication with persistent or recurrent hemothorax with threatening exsanguination. Thoracotomy should be performed through the 4th left interspace. If necessary the sternum may be transected to improve the exposure. Taking care not to injure the phrenic nerve the pericardium is opened widely and the clotted blood is removed. The site of bleeding may not be immediately apparent but may recur with manipulation. The bleeding is controlled by digital pressure if from the ventricle or may be clamped if from the atrium. The defect is then closed with interrupted 3 - 0 atraumatic silk sutures. Care must be taken to avoid including a coronary artery in a suture. In the presence of a lacerated coronary artery an attempt should be made at repair using 5-0 arterial silk. The intrapericardial aorta or pulmonary artery may likewise require repair. The pericardium is loosely approximated to allow drainage and the chest is closed with waterseal drainage.

**Prognosis**

Parmley[5], states that approximately 25% of patients with penetrating cardiac injuries will reach medical care.

In 1909 Peck[4], reported a 64% mortality in 160 cases of cardiac wounds treated by operation. Many of these died of sepsis. In 1945 Blau[6], reported a mortality of 22% in 27 cases. Twenty-one were treated by operation and sixteen lived while six were treated without operation and five survived. He found that, in several who had been operated upon, the bleeding had ceased. This observation led him to believe that these might have been treated by aspiration alone. In 1951 Elkin[9], reported a mortality of 11% in eighteen cases. Seventeen of these were treated by aspiration, antibiotics and blood replacement with only one death. One patient was operated upon and he died.

**Non-penetrating Cardiac Injuries**

Non-penetrating cardiac injuries may result from direct or indirect, compression or deceleration injuries. Indirect injury may result from abdominal compression producing increased intravascular pressure. They are usually associated with severe thoracic cage trauma but may result from minor trauma such as a blow from a fist.

**Pathology**

**Classification of Lesions (according to Parmley[10])**

1) Parietal pericardial hemorrhage.
2) Parietal pericardial lacerations.
3) Hemopericardium
   1. Acute
   2. Organizing.
4) Pericarditis.
5) Cardiac hemorrhage or contusion
   1. Sub-epicardial
   2. Myocardial
   3. Sub-endocardial.
6) Lacerations and contusions.
7) Rupture of the heart.

Pericardial hemorrhage or laceration when present as an isolated lesion is usually of little significance. However, pericardial laceration is usually associated with rupture of the heart and instantaneous death. Hemopericardium may result from rupture of superficial vessels or a coronary artery producing cardiac tamponade. Cardiac contusion is a not uncommon finding. When the myocardium is involved, focal necrosis with late rupture, especially in the second week, may occur. When the endocardium is involved mural thrombosis with distal embolization sometimes follows. Lacerations of valves, papillary muscles and chordae tendinae are usually associated with cardiac rupture but occasionally occur alone with temporary suvival. Rarely, rupture of an atrium is associated with temporary survival.

**Diagnosis and Management**

Cardiac tamponade has the same implications and is treated similarly to when it occurs in penetrating injuries.

Contusions may be suspected because of arrhythmias or electrocardiographic changes suggestive of myocardial infarction. Depending on the severity of the lesion suspected, bed rest is advised for two to four weeks. Although mural thrombosis may complicate contusions, anticoagulants are contraindicated because of the danger of hemorrhage.

Arrhythmias should be treated as in other cardiac disease.

Rarely a ruptured valve or papillary muscle may be repaired utilizing extracorporeal circulation.

**Prognosis**

The prognosis for contusions is good particularly after the first three to four weeks.

### Penetrating Injuries of the Thoracic Aorta

Penetrating injuries of the aorta are usually rapidly fatal due to exsanguination or acute cardiac tamponade. In a post mortem study on 66 cases Parmley[5] found that 19.6% survived the immediate post injury period.

**Historical Note**

Up until 1958 there had been seven cases of penetrating wounds of the thoracic aorta treated successfully. These were all stab wounds. The first case was reported by Dshanelidze[11] of Russia in 1922. Blalock[12], Elkin[13, 14] and Beattie[15] subsequently reported successful repairs. These were all intrapericardial lacerations. In 1958 Perkins and Elchos[16], and Baret et al[17] each reported successful suturing of extrapericardial lacerations of the thoracic aorta.

**Pathology**

The intrapericardial lacerations are controlled by the cardiac tamponade but death may occur quickly by this mechanism. Occasionally the extrapericardial lacerations are controlled temporarily by surrounding structures with the development of a peri-aortic hematoma or false aneurysm. Parmley[5] described five with false aneurysms which all eventually ruptured. The longest survival was 70 days.

**Diagnosis and Management**

The presence of a stab wound in the neck, chest or upper abdomen should make one suspect the diagnosis. Symptoms and signs of cardiac tamponade indicate the necessity for pericardiocentesis. This will be followed by rapid recurrence and will make operative intervention mandatory. In extrapericardial lesions clinical evidence of hemorrhage and rapid mediastinal widening as demonstrated radiologically is an indication for urgent surgical therapy.

**Prognosis**

The few reports of successful repair are indicative of the seriousness of this lesion. Parmley[5] estimates from a post-mortem study that 20% survive the immediate effects and therefore may be amenable to therapy. Parmley[5] also estimates the mortality to be 30% in the presence of surgical intervention. The prognosis may be improved by early recognition and emergency surgical therapy.

### Non-penetrating Injuries of the Thoracic Aorta

Aortic rupture accompanying non-penetrating trauma is usually a result of a decelerating injury sustained in an automobile accident or a fall from a height. In 72 cases reported by Strassman[18] fifty-one occurred in automobile accidents and ten in falls.

**Pathology**

The site of the injury is usually just distal to the left subclavian artery. In 90 cases reviewed by Bradford[19], 47 ruptures occurred just below the origin of the left subclavian artery. Twenty occurred in the ascending aorta and seventeen in the descending aorta. The aortic arch is fixed by its branches and the ligamentum arteriosum so that the mobile descending aorta decelerates at a rate less than the arch and places the strain on this portion of the aorta. The tears are nearly always transverse. When the rupture is complete death is usually instantaneous. However, with rupture of the intima and media, the intact adventitia and surrounding structures may be sufficient to support the aorta at least temporarily. Rupture, dissection with or without rupture, or aneurysm will follow the initial trauma. Rupture may occur anytime from a few hours to several weeks after the injury. There is usually a premonitory slow leak succeeded within a few hours by exsanguination. It is in this interval that the lesion must be recognized if definitive therapy is to be effective.

**Diagnosis**

In all severe chest injuries the lesion should be suspected. Initial chest x-rays are helpful in assessing the development of mediastinal widening. Serial bedside films are indicated. The radiological signs of aortic rupture are: a) widening of the mediastinum with obscuration of the aortic arch followed by b) a vague bulge in the region of the aortic knob. After a few months a well demarcated aneurysm may be demonstrated. Chest pain is characteristically present. Diminished peripheral pulses may indicate distal dissection of the aorta. Pseudocoarctation with proximal hypertension and absent distal pulses has been observed by Malm[20].

**Management**

If a rupture is suspected close clinical and radiological observation is indicated. In the presence of unexplained bleeding, progressive mediastinal widening, or dissection, emergency thoracotomy is necessary. An approach through the left fourth interspace provides access to the commonest site of the lesion. Resection of a segment of the traumatized aorta with prosthetic replacement may be necessary. Several adjuncts have been introduced to reduce the danger to the spinal cord and distal organs, as well as to reduce the proximal hypertension necessarily produced by cross clamping the aorta. These include hypothermia, temporary external shunt from the left subclavian artery to the femoral artery, and the left atrium to femoral artery shunt. The latter is the procedure of choice at the present time.

Post traumatic aneurysms, of the thoracic aorta may be resected using a similar technique. Steinberg[21], in reporting five cases with a history varying from one to twenty-seven years, makes a strong plea for conservative treatment of traumatic aortic

aneurysms. The natural history of traumatic aneurysms of the thoracic aorta is much longer than those of syphilitic or arteriosclerotic origin. In the presence of symptoms such as back pain, dyspnoea or hoarseness, or radiological evidence of enlargement they should be resected. Eiseman[22] advises follow up of traumatic aneurysms at six month intervals with excision if there is evidence of enlargement.

**Prognosis**

Most case reports of traumatic rupture of the aorta are post mortem studies. With early diagnosis and surgical intervention some of these could undoubtedly be salvaged. According to Malm[20] the mortality for resection of thoracic aneurysms is presently about 19% utilizing the left atrium to femoral artery by-pass.

**Summary**

1) Penetrating and non-penetrating injuries of the heart and thoracic aorta have been reviewed.

2) The pathology, management and prognosis of these lesions have been discussed.

3. A plea is made for early recognition and therapy.

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## A Short Note on Emergency Blood Replacement

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Traumatic injuries are frequently complicated by varying degrees of blood loss. This loss may be external or internal—into body tissues or cavities. The physician is then confronted with the problem of restoring the circulating blood volume. These measures may be life-saving, but unless he is aware of the dangers attending this form of therapy, he cannot make a wise decision regarding their use.

The circulatory effects of hemorrhage depend on the volume of blood loss. The loss of 400 to 500 ml. within five to ten minutes from a normal adult usually produces no change in pulse rate or blood pressure. If a volume of 1000 ml. of blood is lost this will usually produce a fall in blood pressure on changing from a lying to an upright position and may result in loss of consciousness. The loss of 1500 - 2000 ml. will result in an increase in heart rate, drop in blood pressure, and the subject will be cold, clammy and dyspnoeic, or in a state described clinically as shock. In anesthetized subjects these changes may be minimal and confined to a small drop in blood pressure and increase in pulse rate.

In such cases the immediate restoration of blood volume is necessary to preserve life or prevent irreversible tissue damage. The choice lies between the synthetic plasma expanders, serum albumin, and whole blood.

Where blood loss has been great it is necessary to attempt to restore the blood volume and at least partially restore the circulating red cell mass. The ideal is to replace approximately as much whole blood as the patient has lost. If time permits, this blood should be cross-matched. If there is an immediate threat to life because of blood loss, then emergency Group O, low titre, Rh negative blood may be used. This should only be used under circumstances of extreme emergency, as it is not without risk. The use of such blood by-passes the cross-match procedure and thus, one of the principal protective measures for avoiding transfusion reactions.

The emergency blood provided is Group-O, cde/cde. The plasma has been screened so that it is known to contain no irregular antibodies and the anti-A and anti-B present is of low titre. Such blood may contain (and frequently does) other antigens such as Kell, Duffy, etc., which are capable of inducing antibody formation in a recipient lacking

these antigens. Further, if the transfused patient already has such antibody as a result of previous transfusions or pregnancy, a transfusion reaction may result. The risk of using emergency blood is thus greater in those patients who have had previous pregnancies or transfusions. It is true that it is better to have a live patient with antibodies than a dead one due to blood loss, but it is best to have a live patient without antibody or transfusion reaction.

The rapid replacement of large amounts of blood introduces certain transfusion hazards. One of the most dramatic is that of air embolism. If blood must be replaced rapidly under pressure, then it is most important that air not be introduced into the blood bottle directly by pump. The blood may be pumped in using the plastic ball valve system on the administration set, or if the blood has been provided in a plastic bag, external pressure may safely be applied to the bag.

Another immediate danger is that of over-transfusion with resultant rapid expansion of the circulating blood volume. Pre-transfusion blood volume determinations can be made rapidly under certain circumstances, using radioactive iodinated serum albumin or red cells tagged with $Cr^{51}$ or $P^{32}$. However, lack of a steady state, inadequate mixing and alterations in small vessel hematocrit tend to make this estimation inaccurate and careful clinical judgment may be the best practical guide to transfusion requirements. Over-transfusion may result in a rapidly progressive acute pulmonary edema, in some cases associated with auricular fibrillation. When blood is being administered rapidly, frequent auscultation of the chest and a close check on the pulse and arterial pressures should be maintained.

Rapid massive transfusion may induce hemorrhagic disorders due to a variety of causes. The cause of some of the coagulation disorders is that of dilution. Stored blood contains a negligible number of platelets and low concentrations of Factor V and AHG (anti-hemophilic globulin) which are not stable on storage. Patients who receive multiple transfusions within a period of a few hours may develop petechiae and spontaneous hemorrhage into the skin, gut and urinary tract. Hemorrhagic phenomena have been noted where blood in excess of five litres has been transfused within a period of 48 hours. In most cases the bleeding is due to platelet deficiency. This should always be considered when hemorrhagic phenomena follows the replacement of large volumes of blood—and can be determined by a platelet count or by assessing the platelet number from the blood smear. This can then be corrected by the infusion of fresh platelet-rich blood or plasma taken into plastic or silicone containers.

When massive multiple transfusion has been used, varying degrees of hemolysis of the donor or recipient cells sometimes occurs. As red cells contain a powerful thromboplastic agent, this may lead to increased thromboplastic activity which may activate the clotting mechanism and lead to consumption of various coagulation factors. This produces increasing thrombocytopenia, and depletion particularly of AHG, Factor V, and fibrinogen.

Much attention in the past has been focussed on the adverse effects of excess citrate administration. The importance of citrate toxicity in adults is not well established. The presence of excess citrate, binding ionized calcium with the production of tetany and cardiac arrest, may be of some importance in patients with liver disease where the rate of citrate removal may be decreased. However, in the presence of normal liver function it is thought that the liver can metabolize the amount of citrate present in blood stored in ACD within a period of five to ten minutes. Citrate toxicity is probably an infrequent danger in massive transfusion and almost never contributes to a bleeding tendency.

The most commonly used plasma substitutes in this area are dextran and polyvinyl pyrollidone. These substances are excellent as a temporary measure in sustaining the patient until blood arrives. The dextran molecules are long chains of glucose units and the average molecular size may vary with different dextran solutions. The size of the molecule will determine its length of stay within the vascular tree. The British product, in common use in this area, has a molecular weight of approximately 200,000. The presence of dextran or polyvinyl pyrollidone causes marked rouleaux formation which may interfere with the interpretation of red cell compatibility tests. Before beginning an infusion of dextran, a blood sample should be obtained from the patient and put away for cross-match procedures in case blood is subsequently required. Following the use of dextran, allergic responses have been noted. These may take the form of flushing of the face and neck, headache, malaise, fever and vomiting. Following the use of large dextran infusions, bleeding may occur and this has been produced following the use of 1500 ml. of dextran—these may be due to rapidly developing thrombocytopenia and hypofibrinogenemia.

In patients where the primary trauma is that of an extensive burn or where a burn complicates blood loss, the replacement of red cell mass must be accompanied by an attempt to restore the circulating plasma volume. The best substance for this purpose is serum albumin. The increase in plasma volume brought about by serum albumin is related to its protein content rather than to the volume infused. Fluid is drawn into the vascular space to restore the osmotic pressure and in this way expands the vascular compartment.

The substances mentioned are valuable therapeutic tools and frequently can save life. However, it is important to realize their limitations and the potential hazards attending their use.

# Abdominal Trauma

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This presentation will be confined to pure abdominal injuries, excluding thoraco-abdominal injuries and those of the genito-urinary tract.

With the increased numbers of traffic accidents, with increased mechanization in industry, with increased numbers of "trigger happy" hunters abdominal injuries present themselves not infrequently

## Classification

Abdominal injuries are usually classified as penetrating and non-penetrating, the latter frequently referred to as blunt trauma. Either type of injury may result in laceration of a solid organ or perforation of a hollow viscus or both. This results in hemorrhage and/or sepsis, both of which combine to yield a high mortality, if treatment is delayed or neglected.

Mortality can be reduced by:

(a) Having a high index of suspicion regarding abdominal injury, especially in the presence of multiple injuries.

(b) Employing prompt and proper resuscitative measures.

(c) Evacuation of such injured patients to an area where definitive surgery can be performed.

## First Aid Treatment

The doctor who first sees these injured patients should attempt to ease pain, combat shock, dress the wound and provide early evacuation. He should observe the following rules:

1. Cover all abdominal wounds to prevent contamination (never probe).
2. Never replace a prolapsed viscus.
3. Initiate shock therapy by using blood or plasma expander.
4. Insert a Levin tube in the stomach.
5. Insert a catheter in the bladder.
6. Oxygen.
7. Antibiotics.
8. Evacuate once the cardio-respiratory system is stabilized.

## Penetrating Injuries

Penetrating wounds usually result from sharp objects such as ice picks, knives, glass, from stabbings or from bullets or other foreign material resulting from gun powder, or similar explosive materials.

In the early months of World War I the general policy of the treatment of abdominal injuries was non-intervention, for two reasons: (a) it was thought that the injured would die anyway, and (b) it was regarded that abdominal surgery at the front lines was impracticable.

Thus when the patients were seen later, most of them had developed peritonitis. Mobile facilities, however, allowed earlier surgery to be done, and by the War's end it was generally agreed that most abdominal injuries should be treated surgically. This was so well remembered in World War II that the fundamental premise in the management of abdominal injuries was that all major wounds were to be explored as soon as resuscitation was received. This was no defeatist attitude, but a positive one. So much so that less than one percent of the casualties treated by the 2nd Auxiliary Surgical Group of the U.S.A. were not operated upon. This practice was continued in the Korean War and apparently was better applied for the mortality rate was only fifty percent of that of World War II in comparable organ damage cases.

These were lessons well learned and are now applied to civilian practice. It is fallacious in treating penetrating wounds of the abdomen to delay early exploration.

While preparation for surgery is under way, resuscitative measures are begun. Patients who respond poorly to such measures have been found to be those with the more serious intra-abdominal injuries.

## Diagnosis

Diagnosis of the injury requires a detailed history of the agent causing the injury, the path of the wounding instrument, the position of the body when wounded, entrance and exit wound determination, etc. Note must be made of any vomiting of blood or passage of blood per rectum. Physical examination of the disrobed patient must be gentle and thorough. Splinting of the abdomen, tenderness, shifting dullness, distension all are evaluated. The presence or absence of peristaltic sounds are extremely important, for normal sounds preclude a peritonitis while absent sounds, although not invariably, connote serious injury. X-ray of the abdomen may reveal an opaque foreign body, and from the site of entrance damage to intervening tissue may be determined. However, such reasoning may be highly inaccurate for a bullet may be deflected by a bony structure. If the findings are inconclusive on examination, observation and re-examination by the same examiner will determine whether surgery is necessary. Negative laparotomies should not be a reflection on the diagnostic acumen of the surgeon.

## Non-Penetrating Injuries

The increased incidence of motor accidents has increased the incidence of non-penetrating injuries of the abdomen. These too often are overlooked because of the frequent association with other more

obvious injuries which tend to distract the examiner. Crushing between a truck and loading platform, blasts (air or water), blows by fists, falling debris, may all be the cause of such injury. A sharp blow is more likely to cause rupture than slow pressure. A distended viscus either with food or gas or feces or urine is more prone to rupture than an empty one. (Napoleon's order to his soldiers to empty their bladders before engaging in combat was a sound one). Fixed viscera near the spine, such as the pancreas, kidneys and duodenum may be injured. Injury to the small bowel usually occurs at its points of fixation at the ligament of Treitz, ileo-cecal valve or mesenteric attachment. The liver and spleen, well protected as they are, are frequently injured in association with fractures of the overlying ribs. Rupture of intra-abdominal, extra-peritoneal visci such as colon, duodenum, bladder may go unrecognized because of lack of intra-peritoneal irritation and therefore close observation of the patient is essential.

The main decision to be made, irrespective of accurately determining the actual lesion, is whether or not immediate surgery is necessary to control bleeding or repair a ruptured viscus. Delay in this vital decision is an important etiological factor in the mortality associated with non-penetrating trauma.

**Diagnosis**

Diagnosis is based upon the history of the accident, subsequent symptoms, physical examination, x-ray and laboratory tests, and abdominal paracentesis.

From the history information will be obtained regarding ingestion of food and alcohol, type of work, type of accident, method of injury, etc. All areas of discomfort, history of vomiting, location of pain, hematuria and so forth, must be ascertained.

During the physical examination associated injuries must always be suspected and sought for, and resuscitative measures should again be begun before any detailed examination is considered. Inspection for abnormalities of respiration, peristalsis, masses, followed by careful palpation for tenderness, guarding rigidity, auscultation for presence or absence of bowel sounds, and rectal examination is a minimum examination. If no conclusion is arrived at, re-examination every fifteen to thirty minutes is mandatory until a decision for or against operation is made.

Laboratory tests include urinalysis to denote the presence of gross microscopic hematuria. Hemoglobin and hematocrit estimations, if only done once, are well nigh useless, for repeated examination may indicate continuing blood loss. Serial blood volume estimations, if they can be done rapidly, are the best indicators of progressive hemorrhage. Serum Amylase estimations should always be done in severe upper abdominal trauma to rule out massive pancreatitis, which is an important contributory factor in the mortality. A leucocystosis of greater than twenty thousand before peritonitis has set in is suggestive of splenic rupture.

X-ray examination of the patient should include flat films of the abdomen in the upright and lying positions to determine the presence of air beneath the diaphragm. Obliteration of psoas shadows by gas or fluid indicates duodenal rupture, retroperitoneal fluid accumulations, etc. Fractures of the lower ribs and pelvis and lumbar spine may also be revealed. Flat films taken one-half hour after intravenous diodrast demonstrating extravasated dye may be helpful in detecting ruptures of the liver (but more important in bladder and kidney injuries). Only positive x-ray findings are of value, for perforations and ruptures of visci may still be present despite the absence of free air in the peritoneal cavity.

Four quadrant abdominal paracentesis has been praised by many and deplored by others. This procedure is useful only when positive results are obtained, and has its greatest value in patients with multiple injuries, especially where the signs and symptoms of intra-abdominal injury are masked by shock, concussion, hemorrhage or acute alcoholism.

If no diagnosis is obtained and re-examination at later intervals still gives equivocal findings, one may obtain guidance from the following dictum: If abdominal pain, tenderness and spasm persist for six hours in a patient known to have suffered severe trauma to the abdomen and under observation, then an exploratory laparotomy is indicated.

A decision to continue conservative management is made because of minimal objective findings and subjective complaints. It is essential to remember, however, that there are a number of conditions that may delay or obscure the signs and symptoms of intra-abdominal injury. These are — shock, intestinal spasm, plugging of a laceration by a clot, omentum or adherence to nearby viscera, delay in the actual rupture and concomitant multiple injuries. At all times conservative management may be only a temporary expedient because of the conditions enumerated and a decision for surgical intervention may be made when changes in the patient are recognized.

**Surgical Treatment**

Wounds of entrance and exit should serve as guides for the placing of incisions, and not a starting point, unless the stab wound has been made by a knife in which case the stab wound may be incorporated in the incision. Incisions are usually placed at the supposed site of the injury, with accommodation for extension. In most instances, except when one is certain that only the spleen is involved (in which case a left subcostal or paramedian approach is utilized), the peritoneal cavity

is entered through a long vertical incision, sometimes running from the costal margin to the pubis. In general, when a surgeon enters the abdominal cavity of such patients his aim is to:

(a) Control all bleeding.
(b) Thoroughly and systematically examine all viscera.
(c) Thoroughly care for all wounds encountered.
(d) Debride all nonviable tissue and remove all accessible foreign bodies.
(e) Drain adequately, when required.

Stab wounds of all the viscera can usually be repaired per primum, except in the case of the spleen, where any injuries which cause a tear or break in the capsule demand splenectomy. This general rule for splenic injury obtains even if bleeding from this organ has ceased, or even if there is subcapsular hemorrhage, in order to prevent the disastrous effects that may ensue from delayed rupture.

Liver injuries result in hemorrhage and bile spillage. Lacerations can be repaired, bleeding vessels and bile ducts, if seen, may be ligated. Loose fragments of liver tissue should be removed. Absorbable hemostatic packs can be used to stop the bleeding, which frequently has ceased by the time laparotomy is performed. Because of the dangers and deaths from secondary hemorrhage at the time of their removal, non-absorbable packs should not be used. Because of the dangers of bile peritonitis and subphrenic abscesses, all liver wounds should be widely drained.

Wounds of the stomach can usually be closed, and it is necessary to open the lesser sac and inspect the posterior aspect of the stomach.

The small bowel and its mesentery must be closely scrutinized from the ligament of Treitz to the ileo-cecal valve, all perforations and injured areas closed, and even segments resected and continuity maintained by end to end anastamosis when required. Only under rare circumstances should exteriorization be necessary.

Duodenal injuries are frequently difficult to detect because of retroperitoneal location. If damage is suspected, wide mobilization is mandatory for its proper inspection. Repair of lacerations here should be meticulously carried out and the area should be well drained.

Occasionally the common bile duct is transected (only rarely by itself) or damaged by blunt trauma. Repair over a T-tube and wide drainage is the treatment of choice. Gallbladder ruptures may be treated by cholecystotomy, but cholecystectomy is preferable.

Despite its posterior situation the pancreas can be injured and should be treated conservatively as an acute pancreatitis. If damage to this organ is noticed at laparotomy many authors recommend choledochostomy, repair of the injured ducts if possible, suturing of the capsule, and wide drainage of the lesser and greater omental sacs and even of the retroperitoneal space.

Because of their retroperitoneal location certain portions of the colon, especially the flexures, require extensive mobilization for examination. There should be no hesitancy whatsoever in doing this especially in bullet or stab wounds, for overlooking such wounds will permit contamination of the peritoneal cavity, by heavily infected contents.

Wounds of the colon have undergone a change in their management. During wartime all colonic wounds were either exteriorized or closed, but always with a diverting colostomy. Recent analysis of civilian injuries has shown that in selected cases, laparotomized early and with minimal contamination, primary closure without colostomy gives satisfactory results. Severe injuries of the cecum and ascending colon are best treated by ileotransverse colostomy rather than exteriorization. Ileostomy should almost never be required.

Extraperitoneal rectal wounding should always be treated by proximal colostomy, repair of the wound if possible, and presacral drainage established through a retro-rectal incision. It is usually unnecessary to remove the coccyx.

**Summary**

Improved results in the management of abdominal trauma can be obtained by vigorous treatment of shock, which is the greatest mortality factor. Measures to combat this feature and some specific treatment features have been outlined.

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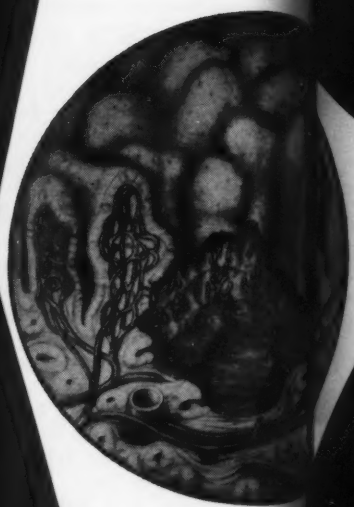
strengthens
fragile capillaries

# in internal bleeding



capillary hemorrhage
in duodenal ulcer

associated with abnormal capillary permeability and fragility in

**peptic ulcer**

**ulcerative colitis**

**chronic nosebleed**

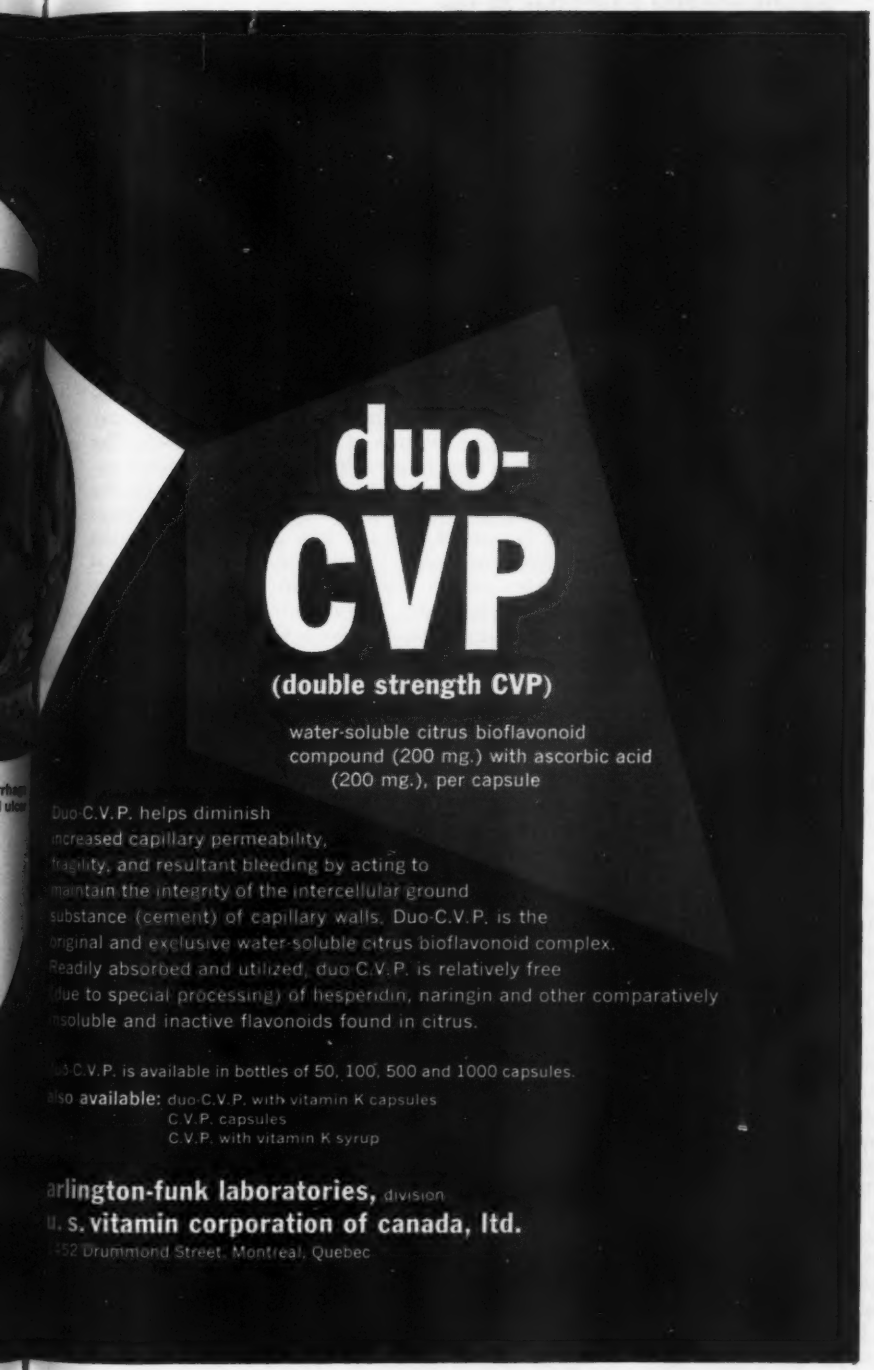
**purpura**
(nonthrombocytopenic)

**hemorrhagic cystitis**

**ecchymoses**

**menorrhagia**

**habitual and threatened abortion**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Duo-C.V.P. helps diminish
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fragility, and resultant bleeding by acting to
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Readily absorbed and utilized, duo C.V.P. is relatively free
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252 Drummond Street, Montreal, Quebec

# Supracondylar Fractures of the Humerus in Children

F. R. Tucker, M.D.

Although this is not a common fracture, it constitutes approximately 60% of the fractures in the region of the elbow-joint in children. It is a difficult and a dangerous fracture. The fracture occurs across the lower end of the humerus in the region of the condyles, and is immediately proximal to the epiphyseal plates of cartilage. The distal fragment may be displaced posteriorly or less commonly in an anterior direction. It is important to distinguish the anterior from the posterior variety because the methods of reduction are diametrically opposite.

In the posterior type of supracondylar fracture, the distal fragment is displaced posteriorly and is often angulated posteriorly and rotated or displaced medially or laterally. If there is a significant displacement of the bony fragment, there is always some accompanying soft tissue damage. The soft tissues most vulnerable to injury are the brachialis muscle, the brachial artery and vein, and the median and radial nerves. The amount of soft tissue damage is usually in relationship to the violence applied and the degree of displacement of the distal fragment. As a result of the fracture and the soft tissue damage, there is bleeding and collection of oedema in the soft parts, particularly in the cubital fossa. The rate of development of swelling is often amazingly fast. The effect of the local distention by these fluids is to further jeopardize the circulation of the part, as well as distend and inflate the soft tissues and limit their longitudinal stretch. This is a factor in making reduction by traction more difficult.

It is the soft tissue damage and the swelling which makes the supracondylar fracture dangerous. It is important for the surgeon to assess the degree of soft tissue damage by a careful examination of the peripheral circulation and the integrity of the important nerves. The circulation is best tested by palpating for the radial pulse and by examining the capillary circulation in the fingers. The absence of the radial pulse is a serious sign of arterial impairment, but the circulation in the fingers is of greater significance. Coldness and lack of return of blood to the capillary bed in the nails after compression signifies occlusion of the arterial circulation. The median nerve can be tested by asking the patient to flex the fingers, or to oppose the thumb to the little finger. Testing sensation in the thumb, index and middle fingers is also valuable, as this region is supplied by the median nerve. The integrity of the radial nerve is easily tested by having the patient extend the wrist or extend the metacarpophalangeal joints. The x-ray examination is then performed, and this completes the investigation.

Treatment of a fracture is usually discussed under the headings of reduction, immobilization, and restoration of function by exercise. In this fracture, however, the methods of reduction and fixation are dependent upon the amount of swelling and the circulatory damage. It is vital to realize that the future of the limb is more dependent upon the soft tissue injury than the bony injury. Should the swelling in the elbow region be minimal, even if there is circulatory embarrassment, one may proceed with the routine methods of reduction. This consists in longitudinal traction which is applied to overcome the shortening and to release any soft tissues which are trapped between the two fragments. When the limb is drawn out to length, it is important at this moment to correct any angulation or rotation of the distal fragment. The fracture is then hyperextended to unlock the fragments and the thumb is shifted distally to the region of the olecranon process and the distal fragment is pushed forward into alignment with the proximal fragment. There are usually sufficient irregularities on the fractured ends to lock the fragments together. The limb is then flexed at the elbow to an angle of about 45° above the right angle. This is the optimum position of the mobilization, but this angle is only permissible if the circulation in hand and fingers is adequate. A supporting bandage of elastocrepe may be applied over the arm, elbow, and forearm to control swelling, and a collar and cuff sling is applied. It is wise to avoid gauze bandage, adhesive tape or plaster casts, as these may produce constriction in the elbow region and effect the peripheral circulation. The position of flexion is the position of stability. Two factors which lead to stability are the interlocking teeth at the fracture site, and the tension on the posterior periosteum and the triceps muscle. It is very important that the patient and the limb be kept under expert observation for at least twenty-four hours, or until such times as any fear regarding circulation has disappeared.

If the arm is already grossly swollen, or if the elbow cannot be placed in a position of stability it is wise to use alternative methods of reduction and fixation. Skin traction may be applied to the limb by the Dunlop method, or skeletal traction may be procured by passing a Steinman Pin to the upper end of the ulna and to suspend the forearm in a counter-balance sling, so that traction is applied and the limb is elevated, and the peripheral circulation can be examined with ease. Either of these methods is reliable and should be continued until all swelling is settled and the circulation has been fully restored. After this time the elbow may be flexed in the optimum position and a collar and cuff sling applied.

A circulatory embarrassment may be evident on first examination, or it may arise subsequently after

manipulation and immobilization. The first variety is frequently improved by reduction of the fracture. The second variety is serious, and demands prompt action. It is important to remove all encircling bandages and casts, to straighten the limb, and if possible to institute some form of traction. In my own experience I have always found this to be sufficient. However, more serious cases have been described and in these it is important that efforts be made to improve the circulation. The cubital fossa and the anterior surface to the forearm should be liberally incised, dividing the skin and deep fascia. Hematoma should be evacuated and the brachial artery should be examined. In children it has been recommended that if the artery is irreparably damaged or crushed that it be excised following ligation. This paradoxical form of treatment is apparently effective by relieving the spasm in the collateral circulation, and thus improving the circulation to the muscles of the forearm and to the distal extremity. Time is of the essence in treatment. Anoxia of the forearm muscles cannot continue for more than three or four hours without permanent damage and resulting fibrosis. Volkmann's contracture is the result of ischemia to the forearm muscles.

The injuries to the radial and median nerve are almost always due to simple contusion and will recover spontaneously. Apart from protective splinting and efforts to retain mobility of the joints no further specific treatment is usually required for these nerve injuries.

There are many possible causes for the ischemia of the limb and the development of Volkmann's contracture. On some occasions the severity of the vascular injury or delay in seeking treatment may have caused irreversible damage to the flexor muscles of the forearm. On the other hand, the ischemic changes may be due to inadequate or improper treatment. It may simplify an understanding of the problem if the main causes of Volkmann's ischemic contracture are listed. These are as follows: (1) Displacement of the fracture. (2) Compression from hematoma and oedema. (3) Compression by excessive flexion of the elbow. (4) Compression from encircling casts or bandages. (5) Arterial injury. It has been mentioned previously that absence of the radial pulse and circulatory changes in the hand are two important signs of ischemia of the limb. It would be wrong to imply that these were the only two or important signs of ischemia or impending circulatory catastrophy. Especially after reduction of the fracture the child's pain should be easily controllable by simple analgesics such as aspirin. Pain that is greater in amount should immediately arouse one's suspicion that the circulation is defective. The use of the five P's is helpful in the recognition of ischemia. These five P's are: pain, pallor (cyanosis), paraesthesia, paralysis and pulselessness. Forearmed with this information, and aware of the seriousness of the circulatory problems the management of this fracture then becomes much simpler, and the frequency of Volkmann's ischemic contracture will be greatly diminished.

Returning to the problem of the fracture itself, the part is immobilized in the optimum position of elbow flexion for three to five weeks. It is usually best in the child to have the limb inside the clothing for protection. If one feels uncertain about the child and its vulnerability to falling, it is quite permissible to place a posterior plaster slab along the arm and forearm as added protection and support. When the fracture is clinically firm, the support can be reduced and the child allowed to resume his own active movements. Passive movements and stretching are contraindicated. The child does not require special physiotherapy. By intuition he appears to know when the time has come to commence movement. One is not always able to obtain exact anatomical reposition of the fractured fragments. Although it is desirable to achieve perfect position, the circulatory problems or the amount of swelling may prevent the accomplishment of this objective. Fortunately for patient and surgeon, an incomplete reduction is still compatible with ultimate perfection of movement and position. The essential requirement is satisfactory alignment. Some slight posterior displacement, lateral or medial shift, are easily corrected by growth and remodelling of the fragments. It is true that, if there is residual posterior displacement, that a bony block will exist to flexion of the elbow. However, nature gradually removes this block by bone resorption, and with continued growth in the young child this bony block becomes displaced further from the joint by the addition of new bone from the epiphyseal plate. One can, therefore, assure the parents that with growth and remodelling the range of movements of the elbow will return to normal in approximately one year's time. As mentioned previously, lateral or medial deviation of the distal fragment must be corrected by manipulation at the time of initial treatment. Nature does not rectify this deformity as she does with the anteroposterior displacements. Should the carrying angle of the elbow be affected to a significant degree, it is better to accept this deformity for the time being, and later to perform a supracondylar osteotomy to correct the resulting deformity.

The anterior type of supracondylar fracture is much simpler to treat, and in my experience has not been accompanied by the circulatory problems. All that is required is longitudinal traction to overcome the shortening and the correction of the lateral or medial displacement and rotation. The limb is then immobilized with the elbow in full extension. A posterior plaster splint is applied to the arm, elbow and forearm to maintain this position. This is one of the very rare occasions in which an elbow-joint is immobilized in extension. In the child one need have no fear about regaining a full range of flexion and a full recovery of function can be expected.

# Farm Injuries

L. C. Bartlett, M.D., F.R.C.S.(C)

Many a city dweller has fond dreams of moving to a quiet countryside, but he fails to realize that farming is one and a half times as dangerous as the average industry. This farm accident problem is national in scope, but the details vary with the locality.

This paper is based on a 10-year review of the Medical Literature and of the Winnipeg Public Library Farm Literature (1948-1958). Such a review is essential since no one person has sufficient experience to cover all aspects of the problem. Statistics are more readily available for the United States than for Canada, a situation which should be remedied.

Industry concentrates on safety in numerous ways—by studying how accidents happen, by research into means of preventing them, by periodic inspections, by education of the workmen and by Workmen's Compensation Board coverage. In industry, good safety records are rewarded and bad ones are penalized. The farmer, on the other hand, has very few of these advantages. Usually he is his own boss, or has a very few men working for him. He is handling machines as powerful as those of industry, over rough ground, each for only a short season of the year, for long hours and sometimes with makeshift repairs.

Farm accidents are no different from other accidents in that they are usually the result of carelessness. The proper solution, therefore, is first to study the records to see how and why these accidents happen, then to publicize the results, in a process of education.

## Incidence of Farm Accidents

There are few Manitoba figures available but in 1958 there were 31 deaths due to farm accidents in Manitoba. There are excellent statistics available for the U.S.A., and although there are differences in methods of farming, a detailed study of these should be of some help.

Although the U.S.A. farm population is only 1/10th the total population, farm deaths were ¼ the total number of accidental deaths in the country. The death rate due to farm accidents is 53 per 100,000 workers per year, or approximately 1 in 2,000 workers each year. To make this figure significant I would like to point out that all other industries together averaged 31 deaths per 100,000 workers per year. The risk in farming is surpassed only by mining with 71, and construction with 55 deaths per 100,000 workers. Because of the larger numbers employed, however, farming produces the greatest total number of accidental deaths.

What of the non-fatal, farm accidents, that is those needing medical care, or entailing loss of half a day or more from work? 85% of these injuries are: (in order) cuts, scratches, sprains and bruises. Farming has a higher rate of disabling injuries than any other major industry.

In an excellent survey of 4,000 farm families over a period of one year, 650 such injuries occurred with 10 deaths, or one injury per six families and one death per 400 families.

**Where do these farm accidents occur?**

In the most comprehensive survey ever made they occurred as follows:

(a) Grouped according to type of work:
- 65% occurred during farm work
- 6% occurred during house work
- 16% occurred during recreation
- 23% occurred at miscellaneous or other unknown jobs.

(b) Grouped according to site:
- 16% occurred in the home
- 22% occurred in the farm yard
- 34% occurred elsewhere on the farm
- 11% occurred on the road or street
- 17% occurred elsewhere or unknown.

(c) Grouped according to age — in one 10 year study the accident death rate for people over 65 was 338 per 100,000; for children under 5, 55 per 100,000; and from ages 5 to 14, 25.5 per 100,000.

That is, the hazard is greatest in the oldest and next in the youngest.

People over 65, although comprising only 6% of the total farm population, had 30% of the accidental deaths.

(d) Grouped according to sex:
75% of the accidental deaths were in males.

(e) Grouped according to size of farms:
Smaller farms had an accident rate 6 times that of the larger ones.

Let us consider a detailed breakdown of each of these figures in turn:

## Farm Work

**Highway**

Although highway accidents are excluded from most series for the sake of clarity, the risk is nevertheless present and must be stressed. The highway is the most dangerous place of all, and in one series in Iowa caused 42% of farm fatalities. 800 of these were auto injuries and 144 tractor injuries.

**Machines**

Machines caused 25 out of 61 farm injuries reported in the Southern Medical Journal. There are two factors involved—the machine and the man.

Although in earlier years the main emphasis in farm machines was on performance, in more recent years, safety has received more attention. But as in the auto industry, this is not always appreciated by the buyer. For example guards have been removed in making repairs and not replaced.

Generally speaking however, the industry is quite safety conscious and must be commended for its continuing efforts and research in this field.

Now consider the man. When the farmer used horses he had to stop to rest the horses and at the same time necessarily rested himself. With machines on the other hand, the farmer now may work extremely long hours and even darkness is no longer a barrier. He often works with little rest and sometimes misses a meal. These factors of course lead to fatigue, and fatigue in turn leads to increased accident risk.

**What Machines are Involved?**

In a series of 186 farm machine injuries reported from the Mayo Clinic, the causes were in this order: Tractor, Corn Picker, Corn Shredder, Being run over by a machine, Buzz saw. These constitute the largest group.

The others were: Thresher, Grain elevator, Feed mill, Combine, Corn binder, Gas engine, Mower, Rope and pulley, Grain binder, Miscellaneous. One third of these accidents resulted in amputation of one or more extremities.

**Tractors**

In a series in Pennsylvania, tractors caused 14% of the farm accidents but 38% of the deaths. Three-wheel tractors were found to be twice as bad as four-wheel tractors. In a second group over a 3 year period 10% of these accidents involved children under 10 years.

In a 3rd group of 19 tractor injuries, 15 were due to tractors overturning. Tractor deaths have been increasing while other machine deaths have been decreasing. In a 4th series of 700 fatal tractor injuries:

55% were due to overturning
16% due to faults with the vehicle
8% run over
5% squeezed against something
3% entanglement of clothes, etc.
13% unspecified.

30% occurred under the age of 20 years and 10% under the age of 5 years (often falling off while riding).

One-third of the tractor accidents occurred while the tractor was on the highway. Considering the short time the tractor is on the highway this implies a tremendous risk.

**Conclusion**

The following are the National Safety rules regarding machines:

1. Always be alert and never take a chance.
2. Stop all machinery before oiling, adjusting, or unclogging.
3. Don't wear floppy or ragged clothing.
4. Always operate tractors at a safe speed.
5. Start smoothly and slow down for turns and rough ground.
6. Avoid operating too close to ditches or embankments.
7. Small children are definitely out of place around machines.
8. Avoid jumping off the machine when stopping.
9. Look both ways and cross highways with care.
10. On the highway obey the signs and rules. Use headlights and taillights after dark.

To these I would like to add another — put machinery in good order before using. Check rusty bolts in seats, check control levers, steps, shields and guards.

**Miscellaneous Hazards of Farm Work**

**Falls**

Falls caused 193 of 575 farm accidents reported from the Mayo Clinic. The mortality rate was 5%. These falls most commonly occurred from machines, but also from other objects in this order: haymows, buildings, stairs, porches, ice, ladder, scaffold, trees, miscellaneous.

The commonest injury was fractures. Three had spinal cord paralysis. Eight slid, fell or jumped and impaled the handle of a pitchfork in the groin area. Three of these died.

In another series of falls, 25% occurred in barns, 22% from hayracks, 11% from buildings, 7% in barnyards, 7% from haystacks, 5% from scaffolds, 4% from ladders, 3% from trees, and 14% from other objects. There is a marked similarity in these two series.

**Other Miscellaneous Farm Hazards**

**Accident Hazards**

Old wells, Creeks, Ponds, Old Foot bridges, Running barefoot, Sickles and scythes, Mower knives, Toothed harrows, Starting machines in gear, Backing up without looking, Carbon Monoxide poisoning.

Silage gas, (a yellowish brown gas which is sometimes found in silos. Exposure for two to five minutes has resulted in severe pneumonia or death).

Fires, (from 1940 to 1957 there has been a steady increase in the number of deaths due to fires. Fires may well constitute a greater smoking hazard than does lung cancer).

School Buses, (in a study of school bus accidents in 37 states it was found that boarding and leaving the bus is 37 times as dangerous as the ride).

Drownings and firearms: keep firearms unloaded and out of reach. Lock shells in a childproof place. Teach children the dangers of firearms.

**Health Hazards on the Farm**

These may be grouped under the headings:

**(a) Animal Diseases Transmissible to Man**

Anthrax, brucellosis, encephalitis, rabies, dysentery, trichinosis, bovine tuberculosis, tularemia,

ringworm, actinomycosis. The organisms of gas gangrene and tetanus may also be deposited even in small cuts. It is advisable that all farm personnel be immunized against tetanus.

**(b) Chemicals**

There is no complete catalogue of the tremendous number of chemicals which are available for farm use but generally they include: Insecticides, Fungicides, Herbicides, Rodenticides, Fumigants, Fertilizers, Bactericidals.

Poisoning may occur from inhalation, swallowing in saliva, food or water, or through the skin.

A committee on toxicology and pesticides of the American Medical Association held a symposium on this topic and recommended:

1. More precautionary labeling.
2. More safety awareness by laymen.
3. More accuracy in identifying the agency on death certificate.

**(c) Poison Ivy, Poison Oak and Other Plants.**

**(d) Sanitation:**

Water supply: the efficiency of the water supply is often taken for granted, but how often is it tested? Contamination may lead to gastrointestinal disease such as typhoid, dysentery or the less serious gastro-enteritis with diarrhoea, which is more apt to affect visitors than the family who usually acquire immunity to it. An excessive quantity of nitrates in the well water may result in methemoglobinemia. A contamination of milk supplies may also produce gastrointestinal disturbances. The septic tank effluent may drain into an area where contamination may result.

**Home Hazards**

Home hazards, of course, are not peculiar to the farm, and in fact are slightly lower in the farm home than in the city home, the city home ranking 16.8 deaths per 100,000 population and the farm home, 13.8. A breakdown of these causes of death is as follows:

Falls — 7.9 per 100,000
Burns and fires — 3.2
Poison — 0.7
Firearms — 0.7
Poison gases — 0.5
Others — 3.5

The commonest poisons are barbiturate sedative drugs, aspirin and petroleum products, particularly kerosene, products for external use, especially those containing lead and arsenic, although caustics such as lye, cresol, ammonia, rat poison, flyspray are frequently involved. Poison gases are usually those from vehicles, from heating and from cooking.

**Age Incidence**

The commonest causes of death are: Under one year, suffocation; from age 1 to 65, burns; and over 65, falls.

An analysis of falls occurring in the home is as follows:

| | |
|---|---|
| Steps and stairs | 37% |
| Over object | 14% |
| Slippery floors | 10% |
| From heights | 8% |
| Wet pavement | 6% |
| Hole in the yard | 4% |
| Loose rug | 2% |
| Bath tub | 2% |
| Others | 7% |

**Precautions**

Where there are elderly persons in the family they should get assistance whenever necessary and should be careful of their footing.

Additional precautions should be taken when elderly people are living in the home. The stairs should be well lighted, clear of obstruction, such as marbles or roller skates, with strong railings and enough head room. Worn or broken steps should be replaced and stair treads or carpets should be used. Rugs should be non-skid. These may be purchased as such or rubber jar rings may be sewn to the underside. Tears in rugs should be mended. Floors should have a minimum of wax and more polish to remove the hazard of slipping. Grease or water should be mopped up immediately. Objects should be picked up from the floor. In bathrooms there should be handles for getting out of the tub, with a non-skid mat on the floor. Soap should never be left in the tub. Walks, steps and porches should be kept free of snow, ice and wet leaves. If they are icy they should be sanded. When necessary a ladder should be used instead of a flimsy substitute. It is well worth the time. The ladder should be placed on a firm footing. Toddlers should sleep in a fall-proof crib and should be protected from stairways.

Firearms and explosives are most dangerous in winter and to children under the age of five.

Burns kill more women than any other household hazard except falls.

Spontaneous combustion. Mops and polishing cloths should not be stored in closets or under stairs. They should be kept in metal containers. Rags which are saturated with turpentine or paint should be burned.

Kerosene or gasoline should not be used for starting or hurrying the fire or for drycleaning. A spark from friction may set off a blast. Only non-inflammable solvents should be used.

Heating systems. Chimneys should be cleaned and stovepipes kept tight and rust-free. Chimney dampers should not be closed completely.

Gas stoves should have guards over the controls where there are small children. The oven door should be opened before lighting as gas may accumulate in the oven. Pot handles should be turned to the back of stoves so that small children cannot reach them.

Matches should be kept in a safe receptacle away from children.

Electric shocks. A coin should never be substituted for a fuse. Electrical equipment should never be touched while in the bathtub or when touching any grounded metal, e.g. pipe, tap or rod. Worn out lamp cords or connections should not be used. All equipment should be kept in good condition.

If the washing machine is not grounded, it should have an insulated motor. Electric irons with thermostatic controls are the safest.

Suffocation. Small infants should not sleep with others. They should sleep on a hard mattress with no pillow. They should have covers which cannot tangle around the throat.

Poisons should be marked as such. The label must always be checked before using medicine. Poisonous medications should be kept in a special locked container.

I should like to submit the following check list for home safety:

1. Are stairs and steps kept free of obstructions and well lighted?
2. Are rugs and floors slip-proof?
3. Are all baby toys free of sharp edges and too large to be swallowed?
4. Are sharp tools, poisons and firearms kept out of the reach of small children?
5. Is baby's bedding and clothing always pinned, buttoned or folded in such a way that smothering is impossible?
6. Are matches, pins and needles kept out of reach of children?
7. Are precautions taken against burns or scalds, particularly to small children in the kitchen and bathroom?
8. Are chimneys, stovepipes and gas connections in good condition?
9. Do you guard against carbon monoxide poisoning and oxygen deprivation in your home and car?
10. Is the electrical equipment throughout your home in safe condition?
11. Is your home customarily free from accumulated trash and litter, particularly in out-of-the-way places?

**Value of Safety Education Program**

A number of communities now have farm safety programs which have accomplished a great deal. For example in Iowa there is an Institute of Agricultural Medicine under the Department of Hygiene and Preventive Medicine at the State University Medical College. This consists of a director, who is qualified in engineering and safety, a social worker and a statistician. The University of Wisconsin has a farm safety office which shows a steady decline in farm accidents over the past 10 years. In Illinois an active safety program reduced cornpicker accidents by two-thirds.

**Conclusions**

What brief conclusions can we draw from this mass of data? I should like to make four, namely:

1. Farming ranks as the third most dangerous of the major industries. It is surpassed only by mining and construction.
2. The greatest farm hazards are: (a) the highway, (b) Inadequate supervision of people over 65 and children, particularly regarding falls and burns, (c) Improper use of machines, especially tractors, (d) Common household hazards.
3. Safety measures adopted so successfully by industry should be modified for and adapted to farming.
4. This can best be done by organization of provincial and local farm safety councils. There is as yet no such organization in Manitoba.

# Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

## Guest Editorial

### Trauma

. . . I withdrew some scales of bone and put in each wound a tent with a large head . . . The tents were anointed with a preparation of yolk of egg, Venice turpentine and a little oil of roses . . . Then I applied great compresses steeped in oxycrate and bandaged him not too tight, that he might breathe easily. Next I drew five basins of blood from his right arm considering his youth and his sanguine temperament . . . His diet was barley water, prunes with sugar, at other times broth . . . these things considered, Gentlemen, no other prognosis is possible, save that he will die in a few days to my great grief.

Ambroise Paré.

In the consideration of trauma the physician has many functions, prevention, treatment, rehabilitation and even witness in Court.

Death and disability, like popular music have attracted the attention of the modern statistician, and in the morbid competition for the number one spot, trauma is on the ascendency. In a broad sense all trauma is avoidable including that associated with armed conflict. In civilian life some form of negligence is always evident. All too frequently the negligence is not on the part of the injured person.

Progressive publicity is paid to the relationship of alcohol and traffic accidents, and there is no longer any reasonable doubt of the tremendous role played by this drug. Less publicity is given to other drugs whose usage carries less social stigma, which are nonetheless equally formidable in association with the use of moving machinery or firearms or automobiles. These along with fatigue play an increasing role as yet unmeasurable in the production of trauma.

There is overwhelming evidence from the motor vehicle authorities that a small percentage of the population causes a relatively large percentage of accidents as repeaters. This phenomenon is true in industrial, farm and home accidents. Again it is not always the negligent individual who is injured. It is to the everlasting credit of the inhabitants of North America that the traffic accident rate has not kept pace with the tremendous increase of automobile population.

The medical profession has made phenomenal strides from the days of Paré in dealing with the results of trauma, and this symposium presents an up to date cross-section of this progress.

Psychiatrists repeatedly probe the personality factors which make up the so-called, "accident prone" individual. It is fairly easy to construct a contrasting profile of a cautious, careful, courteous, considerate non-accident prone individual who obeys the rules of life whether at work or driving even in situations where there is no possible chance of "being caught." Such an individual obeys the speed limit and the stop sign, even though he knows the area is not patrolled at the moment. He obeys the rules because he feels it is morally right to do so.

The medical profession has been accused of a "conspiracy of silence," and this accusation has marginal justification. The medical profession is understandably reluctant to testify that a person was intoxicated at a given time by either drugs or alcohol, even though he knows such to be the case. This reluctance to testify is only in part due to the traditional Doctor-Patient relationship and in part to the reluctance of anyone to testify against a fellow man. Mainly this reluctance can be attributed to the anticipated embarrassment on the witness stand.

". . . you say the accused was intoxicated?"

"Yes."

"On the basis of an ataxic gait, slurred speech, bloodshot eyes, fine nystagmoid movements of the eyes, and the odour resembling alcohol on his person?"

"Yes."

"Doctor is it possible for this picture to be produced by any other condition, say for instance a bump on the head?"

"Yes, but . . ."

"Just answer the questions as asked Doctor."

"Doctor, I have here four bottles, one of which contains alcohol and the other three contain other aromatic liquids. Would you be willing to swear that you can tell me which one is alcohol for certainty?"

"Possibly not."

"That is all Doctor."

No Doctor can afford this embarrassment in front of the then coldly sober patient who may actually be a friend or neighbour. Impersonal evidence such as blood alcohol levels will be a more acceptable approach.

The law enforcement and industrial health agencies may demonstrate the accidents to be directly proportional to the extent of rule enforcement. The psychiatrists may be justifiably convinced that accidents are going to happen as long as human

nature remains what it is. The philosophers, comfortably cogent in their cozy corners, may point out that we should not do too much to counterbalance the algebraic progression of births. Through it all unflinchingly and unswervingly our profession pursues its goal of salvaging the injured and the maimed—for this is our philosophy.

Dwight Parkinson, M.D.,
Chairman Manitoba Chapter,
Royal College Committee on Trauma.

**Editor's Note:**

The mighty flood of submitted material has overflown the bounds of this issue, and now promises to engulf the next one. Looking forward to the forthcoming sequel with pleasant anticipation, let us acknowledge our debt of gratitude to our contributors, and to our Chapter of the Committee on Trauma under the able leadership of Dr. D. Parkinson.

## Book Reviews

**"Resuscitation of the Newborn Infant."** Harold Abramson, Editor, C. V. Mosby Co. Ltd., 1960, Price $10.00.

This volume is actually the report of a committee of anesthesiologists, pediatricians, obstetricians and respiratory physiologists as well as pathologists, who have been working in New York City on the problem of perinatal mortality. It was natural that their interests should centre on the problems of the distressed newborn and the problems of resuscitation. This book is therefore a collaborative effort and for this reason there is a considerable amount of overlap. However, a great deal of up-to-date information having to do with physiology and the clinical problems of the newborn infant is contained in this volume. Contributions of Dr. Virginia Apgar and Dr. Stanley James, both of whom have appeared in Winnipeg in recent months, are especially noteworthy. The book is well illustrated, and should be available in every obstetrical unit. The difficulties faced by the pediatrician in determining the cause of respiratory trouble in the newborn infant are well illustrated by a table of "Factors Predisposing to Fetal Anoxia and Neonatal Asphyxia" which names some 200 causes which need to be considered. This volume will not only prove to be of real help to all personnel working in a maternity unit, but also should serve as an excellent guide for those who are given the responsibility of undergraduate and postgraduate teaching of the problems of resuscitation of the newborn.

—H. M.

**Synopsis of Pathology.** W. A. D. Anderson. P. 414. Illust. 4th ed., The C. V. Mosby Company, St. Louis, Missouri, 1960. Price $9.25.

The basic format of this new edition remains the same. It is well organized and concise, and covers the entire general field of pathology in a very able manner. Much of the text has been altered, but in the carefully arranged eleven chapters on general pathology and the fourteen chapters on systemic pathology a number of new concepts of our changing knowledge of disease have been added. This includes a greater emphasis on viral diseases, endocrine disturbances, and the pathology of certain altered biochemical states as in potassium deficiency and the carcinoid syndrome.

The numerous photographs and references are on the whole very good.

If this synopsis is used for that purpose, it is excellent and is of great value to the under and post-graduate students for a quick review. Although it cannot and was not intended to be a substitute for a standard text book, it can be of considerable value as an easy reference and introduction to most aspects of pathology.

D. W. P.

# Questions and Answers Page

In order to keep the medical profession informed as fully as possible in all matters relating to Association business, medical economics and prepaid medical care, this page welcomes questions pertaining to these fields.

We would like to thank the Swan River Valley Medical Group for their letter to the Editor published in the February issue which so clearly and concisely outlined their thoughts and opinions regarding "Medicare."

We are well pleased with the response to the Economic Committee's request for the profession's views in respect to "Medicare," and in addition the opinions that have been submitted in connection with the Association's brief to the Commission on Medical Education. The two subjects are, of course, closely related.

It is felt that the letter from the Swan River Valley Medical Group expresses the opinion of the majority of the profession in the province whether rural or urban practitioners. We readily agree with this group that "Medicare" is unsatisfactory.

The only consolation that exists at the present time, is that the "Medicare" program was experimental in nature and rather than immediately develop a plan that was comprehensive in all respects from which there could be no retreating, it was, we feel, wisely decided that the program should be started on a restricted scale which would provide for improvement and necessary adjustments when and where experience found necessary.

It has been made quite obvious from the letter published and the many communications received that there certainly are areas that require improvement. We are pleased to note that need for improvement is also recognized by the other participants involved in "Medicare." Top level discussions were held some weeks ago to delve into some of the problems that have been expressed. We do not think that we are letting any secrets out of the bag when we say that the Government is considering our recommendation that in areas where there is a high percentage of "Medicare" patients that payment for in-hospital care of such patients should be arranged with the attending physician.

One of the points that the Commission on Medical Education felt that the Association may wish to enlarge upon, was the economics of payment for patient care in the event of any extension of the "Medicare program." Our thoughts on this matter can be stated quite briefly—we feel that home and office calls and in-hospital patient care should be paid for under any enlarged "Medicare" plan. This is a simple statement to make but there are many problems involved in the practical application of this theory. We do not wish to delve into these matters at the present time as they are points that will appear as specific recommendations to the Commission on Medical Education.

We again wish to thank the Swan River Valley Group for their fine presentation of the facts and for their interest in the current economic problems of medical practice.

# Association Page

**C.M.A. President-Elect**

Sincere congratulations are extended to Dr. M. R. MacCharles who was recently selected as Manitoba nominee for the office of President-Elect of C.M.A.

Dr. MacCharles' deep interest in all medical affairs, particularly his keen knowledge and long personal experience in the field of pre-paid medical care should prove to be of great advantage to the C.M.A. in this era of coping with the ever-increasing pressure for "State Medicine."

In 1962, Manitoba will be hosting the C.M.A. Convention.

**Who Should Pay?**

A rather weighty resolution emerged from the 1960 convention of the Associated Hospitals of Manitoba.

The resolution as presented to the M.M.S. was to the effect that M.M.S. take immediate steps to begin making payments to hospitals for diagnostic services. These diagnostic services are interpreted as meaning x-rays and laboratory examinations done on an out patient basis.

This, of course, would be a new departure for M.M.S. and one that has many implications. It is not a request to be taken lightly. The matter is receiving serious consideration by all parties concerned.

It is assumed that a portion of the pressure for such a request arose in those rural areas where the hospital is the only source of providing x-rays and laboratory examinations.

A method now exists whereby a doctor residing in such an area can bill M.M.S. for these services and then arrange settlement with the hospital. A survey conducted by the Associated Hospitals of Manitoba indicates this type of arrangement is not always workable and the problem concerns the fact that the amount collected for these services through M.M.S. is usually less than the hospital's normal charge.

In many instances the doctor concerned is providing a professional service by taking and reading the x-rays and not being remunerated.

These are not all or the only problems involved, but some of the more obvious.

**Doctor Shortage?**

Information emanating from the Royal College of Physicians and Surgeons indicates that Canada is not producing enough doctors. The report indicates that if the trend continues, the shortage could become serious by 1975 or 1980.

There are differences of opinion, on this question of shortage and it may be that only certain provincial areas are affected. In Manitoba the natural "turn-over" of doctors through death, retirement, and moving to "greener pastures" averages around 46 to 50 per year. With 53 students in the current graduating class, Manitoba may maintain the status quo.

Those who feel there is a shortage suggest that something should be done to encourage greater enrollment in the Medical College and they suggest that perhaps a shortened course and greater financial help may be part of the answer. What are your thoughts?

**Area Tissue Committees**

Progress in this connection can be reported due to the action taken at a joint meeting of the Central and Southern Medical Districts held in January.

These two districts have combined for the purposes of forming an Area Tissue Committee. The following were named to the Committee:

Dr. G. M. Black, Portage la Prairie
Dr. G. H. Hamlin, Portage la Prairie
Dr. T. W. D. Miller, Oakville
Dr. W. H. C. North, Carman
Dr. S. S. Toni, Altona
Dr. H. U. Penner, Winkler, as alternate.

The Committee will select their Chairman and advise the M.M.A. office in order that further steps may be taken in respect to notifying hospitals concerned.

It is expected that other District Societies will follow suit in the very near future.

**Socializing?**

On the 30th of January, the Officers shared an enjoyable evening with the caucus of the Provincial C.C.F. party. The members of the caucus were guests of the Association in the Medical Arts Clubrooms, and following dinner an informal discussion took place, covering many facets of health care and the provision of medical service.

Both groups took full advantage of the off-the-record opportunity to air their thinking on the question of "compulsory medical care." Later in the evening the film "On Call to a Nation," a documentary on the National Health Plan in Great Britain, produced by the B.B.C. was shown.

The general feeling appeared to be that the get-together was a good idea, as it is always beneficial to "talk things over."

**Student Education**

Another successful panel discussion arranged for the 4th year medical students was held on January 10th. This was again sponsored by the Economics

Committee, the object being to acquaint the graduating students with some of the economic problems involved in commencing practice. The theme of the panel discussion was "How to Set Up a Practice."

Dr. J. C. Menzies and Dr. M. K. Grace each presented an excellent outline of the requirements, problems and points to inquire into, with regard to rural and urban practice respectively.

Mr. Nitikman, a Chartered Accountant, gave a down to earth talk on accounting practices, income tax requirements and a suggested insurance program for the young doctor just commencing practice. Mr. Brown of the Bank of Montreal rounded out the program by outlining facilities that Chartered Banks in Canada are able to provide. His most welcome words were to the effect that banks consider members of the medical profession A-1 credit risks!

By the interest shown it is obvious that this type of discussion should continue.

**Speech and Hearing Therapy Act**

The Association was asked for its views in connection with the proposed Speech and Hearing Therapy Act which would incorporate the Speech and Hearing Therapists of the province.

The E.E.N.T. Section in their recommendation to the Association indicate that they agree in substance with the incorporation of a Speech and Hearing Therapist Association. It is felt this Act would exclude unqualified and untrained personnel from attempting this type of therapy and would protect the public from improper therapy as all those included in the Asscoiation would be well-qualified personnel.

It is also suggested that there is a definite need for highly trained personnel in this field.

**C.M.A. Questionnaire**

Following the announcement of the establishment of a Royal Commission on Health Service, the C.M.A. released information obtained through a national survey on Health Insurance. The survey was made in 1960 and included all members of the C.M.A.

It is very interesting to note that by a wide and decisive margin, the profession wishes to have the Association (on a federal or provincial basis) negotiate a plan with Government, if Government indicated its intention to start a medical care plan to cover all residents.

Of the 10,000 doctors who replied to the questionnaire, 9,000 agreed this action should be taken.

Another interesting point brought out by the survey is the fact that over 8,000 doctors felt that some form of tax-supported medical care program was either probable in the foreseeable future or inevitable.

Now that the shadow is upon us, it is hoped that we can co-ordinate our efforts and thinking as well as we can predict the future.

**Joint Committee**

The Associated Hospitals of Manitoba and the M.M.A. Joint Committee on Hospital Relations have planned monthly meetings to cope with the variety of subjects on their agenda.

The value of discussion in respect to items of the following nature is immeasurable:

Area Tissue Committees and the role played by the local hospitals in the areas served by the Committees.

The question of remuneration to G.P.s at rural hospitals interpreting x-rays and the possible split of x-rays and laboratory fees into a professional and technical component.

Itinerant surgery—its pros and cons.

Establishment of health services for hospital staff members.

The matter of internes' salaries and the numbers required.

A special joint Committee to provide assistance to small hospitals in respect to medical records.

The Chairman of the Hospital Relations Committee would welcome members' advice on these or other items that may arise through your hospital affiliation.

**Your Ideas**

Suggestions, comments, constructive criticisms, questions, are always welcome. Please direct your remarks to the Review and they will be dealt with in the appropriate column, i.e. Letters to the Editor, Question and Answer Page, Association Page.

**More About Socialized Medicine**

This seems to be a popular subject for discussion and debate. On Monday, February 6th, the Medical Students Association held a panel discussion on socialized medicine, which lived up to the advertising it had received.

Our congratulations to the Medical Students Association for arranging such a well-balanced program and their choice of panelists. Our congratulations also to the Chairman who stole the show at times with his quick and ready wit.

The Chairman introduced the following panelists:

Mr. A. Mackling — C.C.F.
Dr. J. P. Gemmill, U. of M. Faculty of Medicine
Mr. Stan Roberts, M.L.A. — Liberal
Dr. L. R. Rabson, Manitoba Medical Association
Mr. J. A. Christianson, M.L.A.—
Progressive Conservative

and indicated that each was allowed a ten minute initial discussion, following which rebuttal was permitted between them, and at the conclusion, questions were invited from the floor.

Dr. Rabson was named to open the discussion and commenced with a clear outline as to the thinking of the medical profession concerning compulsory medical coverage.

He mentioned the C.M.A. statement on Medical Services Insurance, enlarging on the fact that it was the general opinion of the profession that some type of medical insurance coverage was necessary not only for the indigent but also for the para-indigent groups and that such services should be supported all or in part by the Government. It was his theory that those individuals who can afford to pay their own medical costs should be allowed to do so and select the carrier of their choice. He pointed out the part that M.M.S. now plays in the field of prepaid medical coverage and suggested that M.M.S. be the vehicle through which indigent and para-indigent groups could be underwritten.

Dr. Gemmill followed and his remarks centered on the problems that arise through compulsory prepaid schemes, from the point of view of teaching and the effects on the supply of doctors and the quality of medical care.

Mr. Christiansen suggested that government should not provide those things that individuals can provide for themselves and although he did not mention any medical program that the Conservatives may be developing he mentioned the initiation of the "Medicare" program and the part now played by the medical profession and the M.M.S. in "Medicare," and also mentioned the fact that M.M.S. may be the agency to provide medical services for those unable to pay their own way. He indicated that there were at least 81,000 people over the age of 65 in Manitoba and many of these may require assistance as far as payment for medical care is concerned.

Mr. Roberts refreshed the audience's memory regarding the recent Liberal announcement of an overall medical insurance program for Canadian citizens. Without delving too deeply into the mechanics of the program he mentioned it was based on an income tax formula. He indicated that the Liberal Party Policy Committee was now formulating a proposal which would be available in a few months' time.

Mr. Mackling suggested that he had been invited as a controversial element and he did not intend to disappoint the audience or the other panelists. A few remarks later there was indication that Mr. Mackling had succeeded in his intention. It was quite difficult to absorb his many references, statements reported to have been made by members of the profession across Canada. These references were read so quickly that one imagined Mr. Mackling wished to have his money's worth in the ten minutes allotted. In brief the C.C.F. advocates complete socialized medicine in Canada, the interpretation of socialized medicine including coverage for hospital, medical, dental, optical care and services rendered by para-medical groups. There is no recollection as to an elaboration on financing such a program.

The rebuttal period was most interesting, developing into a debate between Dr. Rabson and Mr. Mackling.

Judging by the applause, the 200 guests (approximately) obviously enjoyed the program and appeared to agree with Dr. Rabson's theories on socialized medicine and his proposals to assist those unable to meet the cost of medical care.

R. P. H. S.



## Central District Medical Society

A meeting of the Central District Medical Society was held at the Manitoba School, Portage la Prairie, on Wednesday, January 18th, 1961.

Present were: Drs. G. Lowther, President; G. H. Hamlin, Secretary-Treasurer; H. S. Atkinson, G. M. Black, R. Gibson, A. J. Grehan, L. P. Millar, T. A. Millar, M. S. Neave, D. W. Rae, J. C. Rennie, D. Steward and C. M. Thomas of Portage la Prairie; W. H. C. North, Carman; H. L. McNicol, Flin Flon; J. H. More, Gladstone; J. C. Menzies, Morden; T. W. D. Miller, Oakville; P. L'Heureux, St. Boniface; M. G. Saunders, St. James; D. W. Penner, St. Vital; H. U. Penner, Winkler; F. H. Burgoyne and M. T. Macfarland, Winnipeg.

The meeting opened with some discussion of Professional Standards Committee (Tissue Committee) in which Drs. Lowther, L'Heureux, Macfarland, McNicol (M.M.A. President) and D. W. Penner, participated. This was postponed until the evening session.

The scientific portion of the meeting was a demonstration of the Electroencephalography facility with a lecture by Dr. M. G. Saunders on E.E.G. Interpretation illustrated by blackboard and slides.

The reception and dinner held at Portage Hotel were well attended.

At the combined business session which followed, Dr. F. H. Burgoyne and three representatives from Southern District Medical Society joined the discussion of Professional Standards Committee (Tissue Committee). Copies of Manitoba Regulation 51/60 and draft copy of proposed M.M.A. directions were distributed.

A resolution proposed by Dr. J. H. More, Gladstone, seconded by Dr. J. C. Menzies, Morden, and carried without dissent, was that Area Tissue Committee No. 2 be composed of Drs. G. M. Black, G. H. Hamlin, Portage la Prairie; T. W. D. Miller, Oakville; W. H. C. North, Carman; S. S. Toni, Altona; H. U. Penner, Winkler (Alternate), and a Pathologist if possible. The Committee will select a Chairman whose name will be sent to M.M.A.

M. T. M.

## Winnipeg Medical Society

Dean L. G. Bell has informed the Winnipeg Medical Society that the University of Manitoba proposes to undertake a new research program concerning the incidence of multiple sclerosis in Greater Winnipeg. Dr. Kurland and members of his staff will be conducting the study.

It will be remembered that 10 years ago a similar survey was carried out which has now come to be regarded as a classic study in the field of population surveys. A new research program is proposed for the following reasons:

1. At the time of the initial studies, Dr. Mathers and Dr. Kurland recognized the possible limitations in the methodology and recommended a re-appraisal of all cases at a later date. Such a re-appraisal offers a unique opportunity to clarify elements in the natural history of multiple sclerosis, to improve still further the methodology of population surveys and to evaluate the accuracy of earlier conclusions on incidence, prevalence and distribution of cases within our city.

2. A resurvey for multiple sclerosis in Winnipeg for the past decade will provide current incidence, prevalence and mortality statistics; comparison of these data with those of the earlier study will help to determine whether or not there has been a real increase in the incidence of this disease.

3. A new effort will be made to compare the Winnipeg multiple sclerosis population with a representative control population in the community to discern any element which predisposes some residents to the disease without apparent reason and to otherwise clarify the etiology of this disease.

The Winnipeg Medical Society on behalf of its members, wishes to heartily endorse this new survey and takes this opportunity to ask that all members give their support where at all possible.

### Report of Nominating Committee

The following report was presented to and accepted by the Council of the Winnipeg Medical Society on February 13, 1961. Prior to that time all candidates agreed to allow their names to be placed on the ballot for election at the Annual Meeting in 1961. The Nominating Committee is pleased to present the following report:

Vice-President:
Dr. Murray Campbell
Dr. A. B. Houston

Secretary:
Dr. J. R. Mitchell
Dr. Edwin A. Osberg

Treasurer:
Dr. John Maclean
Dr. Jean McFarlane

Trustee:
Dr. J. Swan

Honorary Life Members:
Dr. A. M. Goodwin
Dr. Morley Elliott

# Obituaries

### Dr. James Pullar

Dr. James Pullar, 91, died in Deer Lodge Hospital, Winnipeg, on February 9th, 1961. Born in Scotland, he came to Winnipeg in 1880, obtained an Arts degree from Manitoba College and his medical degree from Manitoba Medical College in 1897. He served in France with No. 4 Canadian Casualty Clearing Station in the First World War. He practised in Winnipeg for many years and in his early life was interested in baseball and later in art; painting many pictures.

☆

### Dr. Fred Todd Cadham

Dr. Fred Todd Cadham, 79, died suddenly at his home in Winnipeg on January 29th. Through his uncle, Dr. J. O. Todd, he became associated at an early age with the eminent surgeon, Dr. A. H. Ferguson and this probably determined his choice of career. He graduated in Arts from Manitoba College in 1901 and in Medicine from Manitoba Medical College in 1905, became assistant to Dr. Gordon Bell in the Provincial Laboratory and on Dr. Bell's death was Director for almost thirty-five years. He was appointed Professor of Bacteriology and Head of the Department in 1928 and retired in 1952, being made Professor Emeritus. For 48 years he was a member of the Manitoba Board of Health.

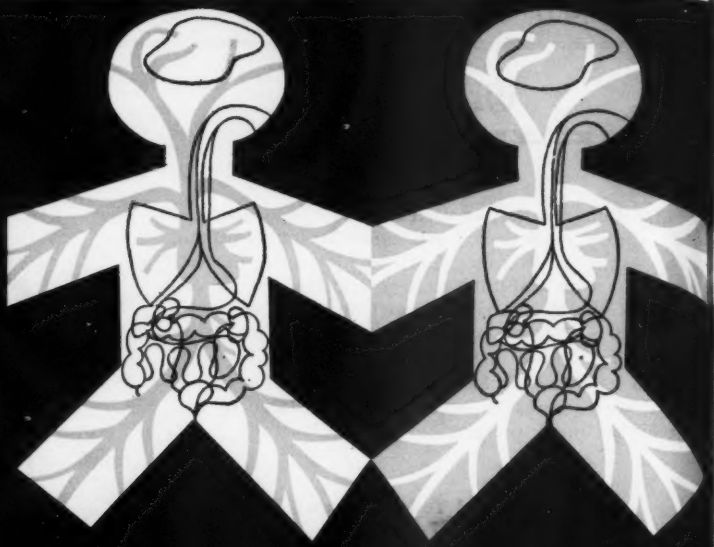
His father was the contractor for the first portion of the present Medical College and Fred may have assisted in the plans of the building. He was a lover of wild life, a keen shot and a natural athlete. He was a member of the Winnipeg Victoria Hockey team which competed in the finals for the Stanley Cup in 1903 against the Montreal Victorias.

In 1916 he went overseas with the rank of major in No. 4 Canadian Casualty Clearing Station and served in France until invalided home in 1918.

As a bacteriologist and immunologist prior to the antibiotic era, he was especially interested in methods of fighting infection by maintaining and augmenting the natural defensive forces of the patient. He was a Fellow of the Royal College of Physicians of Canada, a Governor of the American College of Physicians, a chairman of the laboratory section of the Canadian Public Health Association, and a Senior Member of the Canadian Medical Association.

In 1958 the University of Manitoba granted him the LL.D. degree honoris causa.

His wife died in September 1960 but he is survived by two daughters and a son, Dr. Roper G. Cadham, M.O.H., Winnipeg and Assistant Professor and head of the department of social and preventive medicine in the University of Manitoba.



# high antibiotic concentrations at the site of infection with Cosa-Terramycin

## largest distribution volume

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## high concentrations at the site of infection

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**references:**


1. Kunin, Dornbusch and Maxwell, Finland Journal of Clinical Investigation, November 1959.
2. Spitzy and Hitzenberger Antibiotics Annual, 1957-1958
3. Garrod and Waterworth Antibiotics Annual, 1959-1960

# Social News

Reported by K. Borthwick-Leslie, M.D.

Dr. Bill McTavish maintained the family reputation in grand style in the "Big" Bonspiel. Tough luck in the finals though.

☆

Jimmie Mitchell my "sports" sleuth let me down on the details of the highly successful Bonspiel — courtesy of Elliot Marion Co. in January. Dr. T. K. Goodhand's rink came out on top of the heap with flying colors.

☆

From all reports — though I couldn't make it myself, not being in a party mood these days — the Valentine Party of the General Practitioners was also a huge success. Congratulations and "thank you" to the hard working members in charge of arrangements.

☆

Dr. A. H. Khazei, a native of Iran, who has been doing post graduatework in Surgery at the Winnipeg General Hospital, was chosen as a member of the Canadian medical team for the Red Cross assignments in the Congo Republic. Dr. Khazei, who speaks English, Iranian and French, will join the other members of the team and work out of Leopoldville. Dr. L. G. Sarkin of Montreal is the senior head of the group. The assignment sounds most instructive and interesting. For the sake of all the world let us hope not **too** interesting.

☆

Dr. J. C. Graham, 428 Medical Arts Building, announces that his Surgical practice will now include Thoracic Cardiovascular and General Surgery.

Dr. J. M. Trainor announces the opening of his practice, Clinical Pathology, at Room 1 Medical Arts.

☆

Dr. and Mrs. Neil Margolis proudly announce the birth of a son, February 19, 1961.

☆

Dr. and Mrs. E. W. Stringham welcomed James Andrew Quincy, February 5, 1961.

☆

Dr. and Mrs. G. A. Holman announce the birth of a son, February 2nd at the University Hospital, Saskatoon.

☆

Dr. and Mrs. J. F. Sigurdson announce the arrival of Helen Margaret, February 12th, born in Philadelphia, Pennsylvania.

☆

Dr. and Mrs. Jack Olin proudly announce the birth of their son, Jeffery Frederick, in Toronto General Hospital, brother for Cindy, Shayne and Judy.

☆

Dr. and Mrs. Kenneth McRae announce the arrival of their chosen son, Bruce Allen.

☆

How about some real scandal in the profession for your "Mrs. Winchell"?

# Doctors' Bonspiel

## Calgary - - March 16th and 17th, 1961

The fifth annual Medical Interprovincial Bonspiel will be held at the Glencoe and Meadowlark Curling Clubs in Calgary on March 16-17, 1961.

This Bonspiel is open to medical curlers across the country. Although entries in past years have come mainly from Saskatchewan and Alberta, it is hoped to eventually attract medical curlers from every province in the Dominion.

Edmonton, Saskatoon, Lethbridge and Regina have previously been host to this curl-fest. The highly successful 1960 event was played at the Curlodrome Rink in Regina. Twenty-six entries went to the post. The Dr. A. F. Anderson Trophy, the primary event, was won by the Lloyd Grisdale rink of Edmonton.

There will be four competitions at the Calgary Bonspiel and a host of attractive prizes, and of course a number of social events have been planned for the lady visitors. It is hoped that a number will stay over to enjoy an outing to Banff on Saturday, March 18th. So remember and make it a date — it's the Stampede City in March.

Entries may be sent to Dr. Tom Cranston at 214 - 6th Avenue, S.W., or Dr. Eric Dobbs at 724 Madison Avenue, S.W., before March 8th, 1961. A registration party will be held at the Palliser Hotel from 8 to 11 p.m. March 15th, 1961.

| MIDNIGHT SNACKS for ICE-BOX RAIDERS | | | |
|---|---|---|---|
| Cold potato | ½ medium | 65 | 1.5 |
| Chicken leg | | 88 | 15.0 |
| Milk | 7 oz. glass | 140 | 7.0 |
| Mouthful of roast | ½" × 2" × 3" | 130 | 11.0 |
| Piece of cheese | ¼" × 2" × 3" | 120 | 7.2 |
| Left-over beans | ½ cup | 105 | 6.6 |
| Brownie | 2" × 1¾" × ¾" | 100 | 3.9 |
| Cream-puff | | 150 | 5.7 |
| **SWEETS** | | | |
| Ice cream | | | |
| Plain vanilla | | 150 | 4.0 |
| Chocolate and other | | 150 | 4.5 |
| Milk sherbet | | 130 | 4.0 |
| Sundaes, small chocolate | | 300 | 10.0 |
| Ice cream sodas, chocol | | 270 | 3.5 |
| **CA** | | Calorie Count | Protein Gm. |
| Chocolate bars, 5c size | | | |
| Plain | | 220 | 2.0 |
| With nuts | | 255 | 5.0 |
| Chocolate covered bar | | 270 | 5.0 |
| Chocolate cream, bonbo | | 100 | 0.6 |
| Caramels | | | |
| Plain | | 55 | 0.5 |
| Chocolate nut caram | | 60 | 0.8 |
| **DESSERTS** | | | |
| Pie | | | |
| Fruit—apple, etc. | | 560 | 5.5 |
| Custard | 1/6 pie 1 average serving | 360 | 8.5 |
| Lemon meringue | 1/6 pie 1 average serving | 470 | 6.0 |
| Pumpkin pie with whipped cream | 1/6 pie 1 average serving | 460 | 8.3 |


# ...the proof of the Patrician "200" is in the radiograph!

**When you choose x-ray for private practice,** look at performance as well as the price tag. "Economy" that is gained by short-cuts in table design or a reduction in power may mean slow exposures, blurred radiographs and repeated retakes. General Electric's Patrician "200" combination is designed with adequate power for private practice — a full 200 ma to stop anatomical movement sharply and clearly. Many other features found in larger installations are engineered into the Patrician: 81" table, independent tubestand, shutter limiting and automatic tube protection, to name just a few. And, considering its *uncompromising* G-E quality, this Patrician "package" is remarkably low priced.

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DEPARTMENT OF HEALTH & PUBLIC WELFARE

# COMMUNICABLE DISEASE PICTURE

**North of 53 District**

1 case of bacillary dysentery was reported.

**Northwestern District**

One case of infectious hepatitis was reported.

**Northern District**

Nil.

**Brandon District**

1 case of infectious hepatitis, 5 cases scarlatina was reported.

**Central District**

1 case of infectious hepatitis was reported.

**Winnipeg District**

Reports were received for 14 cases of infectious hepatitis; 1 case of bacillary dysentery; 4 cases of scarlatina.

**Southern District**

Late reports were received of 38 cases of bacillary dysentery which occurred in an institution in Selkirk during December, 1960.

**LIST OF DEATHS FROM COMMUNICABLE DISEASES**

**January, 1961**

URBAN: Cancer, 59; Diarrhoea & Enteritis, 1; Pneumonia Lobar (490), 8; Pneumonias (other forms), 29; Tuberculosis, 1; Bacillary Dysentery, 1; Chickenpox, 1. Other deaths under 1 year, 13. Other deaths over 1 year, 267. Stillbirths, 16. Total, 396.

RURAL: Cancer, 16; Diarrhoea & Enteritis, 1; Pneumonia Lobar (490), 1; Pneumonias (other forms), 8; Tuberculosis, 1; Bacillary Dysentery, 1; Chickenpox, 1. Other deaths under 1 year, 5. Other deaths over 1 year, 83. Stillbirths, 5. Total, 122.

INDIANS: Nil.

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## National Heart Foundation

The appointment of Dr. John B. Armstrong as Executive Director of the National Heart Foundation of Canada was announced today by the Right Honourable Louis St. Laurent, Foundation President.

Dr. Armstrong is a graduate of the University of Toronto Faculty of Medicine, and has done cardiovascular research in Canada, Britain and the United States for over twenty years. He will be responsible for the administrative operation of the Foundation as well as continuing his role as Medical Director, a position he has filled since 1957.

The National Heart Foundation is the federated head of Provincial Heart Foundations in B.C., Alberta, Saskatchewan, Manitoba, Ontario, Quebec and the Atlantic provinces. The Foundations support a nation-wide heart research program which is financed by the annual Canadian Heart Fund now in progress.